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THE LOCAL IMPLANTATION OF SULFANILAMIDE IN COMPOUND FRACTURES

A PRELIMINARY REPORT

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HISTORICAL

THE principle of surgical excision of devitalized and contaminated tissue from traumatic wounds was first recognized and followed by Petit[1] in the early part of the eighteenth century. Larry,[2] surgeon to Napoleon, a century later again called attention to this principle, but even after careful débridement in compound fractures "hospital gangrene" occurred so frequently that amputation remained the prevailing practice. Lister,[3] in 1867, published his first paper on the principle of antiseptic surgery, reporting in detail eleven cases of compound fractures which he had treated with carbolic acid packs. Lister's selection of compound fractures as the proving ground for the idea of antisepsis which Pastëur's work had given him testifies to the temerity of the man, for open fractures still remain the most common traumatic wounds to develop severe infection.

The development of aseptic surgery which followed Lister's work and the better understanding of wound infections gained from the discoveries of bacteriology resulted in progress along these two fundamental principles of treatment of contaminated wounds. Aseptic surgery allowed the effective reduction of the number of bacteria present in a wound and the removal of their best culture media by excision of contaminated and devitalized tissue. Antiseptic agents were developed in abundance, but all suffered from the same defect as Lister's carbolic acid; for, if capable of injuring a pathogenic organism, they were even more capable

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of injuring normal tissue. This severely limited their use as it soon became evident that even the strongest were incapable of complete sterilization of a wound; hence, all that resulted was a yet contaminated, now further devitalized wound. The introduction of neutral hypochlorite of soda by Dakin and its use by Carrel and Dehelly[4] in war wounds is perhaps an exception. Yet it must be used with meticulous care if its irritating propensity is not to outweigh its bacteriostatic effect. Furthermore, technical difficulties make it almost impossible to treat both the fracture and the compound wound simultaneously using Dakin's solution. For this reason its employment has been limited in compound fractures.

TABLE I*

FREQUENCY OF VARIOUS FRACTURES

METHODS OF TREATMENT AND INCIDENCE OF INFECTION

METHOD OF R	TIBIAE FIBULAE		RADII, ULNAE		PHALANGES METACARPALS		FEMORA		HUMERI	
	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%
	17	54.8	7	22.6	4	12.9	1	3.2	2	6.5
Primary suture	15	88.2	7	100.0	3	75.0	1	100.0	2	100.0
Open wound	1	5.9			1	25.0				
Dakin's	1	5.9								
Cast	12	70.6	7	100.0	3	75.0			1	50.0
Traction	2	11.8					1	100.0	1	50.0
Amputation to control infection	3	17.6								
Traumatic amputation	1	5.9			1	25.0				
Gas gangrene	1	5.9								
Infection	4	23.5	1	14.3	1	25.0	1	100.0	1	50.0

Summary: 31 open fractures, 8 became infected } Incidence of infection, 25.8%

*Detailed analysis of the incidence of the various open fractures, their treatment as to method and the incidence of infection occurring in 1932. Thirty-one fractures were treated with an average elapsed time of two hours and forty minutes from time of fracture to beginning of débridement. The average age of the group was 35.8 years. No deaths occurred.

PRESENT STATUS OF TREATMENT

Because of the limitations inherent in all chemical bacteriostatic agents, therapy has improved only with development of mechanical means of preventing infection; that is, débridement, immobilization, postural control of swelling, and stimulation of circulation. These

TABLE II

ANALYSIS OF TREATMENT AND RESULTANT INFECTION FOR 1932

METHOD OF R	PRIMARY SUTURE	OPEN WOUND	DAKIN'S	CAST	TRACTION
No. of cases	28	3	1	23	4
Cases (%)	90.3	9.6	3.2	74.2	12.9
No. of infections	5	3	1	4	2
Infections (%)	17.8	100	100	17.4	50

TABLE III

FREQUENCY OF VARIOUS FRACTURES

METHODS OF TREATMENT AND INCIDENCE OF INFECTION

METHOD OF R	FIBULAE TIBIAE		RADII, ULNAE		PHALANGES METACARPALS		FEMORA		HUMERI	
	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%
	21	52.5	7	17.5	5	12.5	3	7.5	4	10
Primary suture	17	80.9	6	85.7	4	80.0	2	66.6	3	75
Open wound	3	14.3	1	14.2	1	20.0	1	33.3	1	25
Dakin's	1	4.7								
Cast	5	23.8	6	85.7	4	80.0			2	50
Traction	12	57.1					2	66.6	2	50
Amputation to control infection			1	16.7						
Traumatic amputation	1	4.7			1	20.0	1	33.3		
Gas gangrene			4	57.1	1	20.0				
Infection	5	23.8	3	43.0	2	40.0			1	25

Summary: 40 open fractures, 11 became infected } Incidence of infection, 27.5%

Detailed analysis of the incidence of the various open fractures, their treatment as to method, and the incidence of infection occurring in 1937. Forty fractures were treated with an average elapsed time of 3 hours and 15 minutes from time of fracture to beginning of débridement. The average age of the group was 41.6 years. Five deaths occurred, all within 72 hours, 4 as result of head injury, one from rupture of the liver, giving a mortality of 12.5%. All came to autopsy.

measures, together with tetanus and gas gangrene antitoxin, have given excellent results as compared with the dismal situation of Lister's time. Yet, the incidence of severe wound infections following compound fractures remains in the neighborhood of 10 per cent under ideal circumstances and in the best hands. Böhler[5] reports 8.66 per cent severe wound infections in 127 cases of open fractures. He quotes 14 per cent for Koch in 213 cases; 8.6 per cent for Ehalt in 127 cases; and 17.3 per cent for Schmidt in 116 cases. American authors report approximately the same figures. Ritter[2] reports 9 per cent infection; Foster,[6] 15.7 per cent. Poyner,[7] dealing with industrial fractures treated under ideal conditions, reports only 6.3 per cent in 269 cases. The remarkably low incidence reported by Poyner is explained by the fact that from the moment of fracture these cases received expert care by trained first-aid men. Definitive treatment was expert and early, and few were "street wounds."

Municipal hospitals in America, dealing largely with "street wounds" sustained by direct trauma and produced by high speed projectiles

TABLE IV

ANALYSIS OF TREATMENT AND RESULTANT INFECTION FOR 1937

METHOD OF R	PRIMARY SUTURE	OPEN WOUND	DAKIN'S	CAST	TRACTION
No. of cases	32	7	1	17	16
Cases (%)	80	17.5	2.5	42.5	40
No. of infections	8	3	1	7	5
Infections (%)	25	42.8	100.0	41.2	31.2

(automobiles), cannot approximate results such as Poyner's or Böhler's by current methods of treatment. A survey of the records for 1932 embracing 32 compound fractures treated at the Minneapolis General Hospital reveals 25.8 per cent infection. Five years later, in 1937, 40 open fractures resulted in 27.5 per cent infection. These figures include all in which a purulent discharge developed and are not limited to those showing frank osteomyelitis. Yet, examination of these statistics, Tables I to VIII, shows 5 cases of gas gangrene in 1937 and 1 amputation to control infection. In 1932 3 amputations were necessary to control infection, and 1 case of gas gangrene occurred. In both series prophylactic serum was routinely used. In the first three months of 1938, 22 open fractures were treated by us in exactly the same manner as the cases to follow except that sulfanilamide was not used. Infection occurred in 27.2 per cent and there was 1 case of gas gangrene which came to amputation.

This percentage of infection is high; however, when one reviews the material treated at this hospital, the primary healing of over 70 per cent without any evidence of infection seems a triumph. Here we are not dealing with well-fed, employed workmen in good health, but with those on relief, many of whom find themselves there because of pre-existing debilitating diseases. Many are alcoholics and the incidence of delirium tremens is high. Practically all are unwashed and wearing badly soiled clothing. Tabes dorsalis is common and serious concomitant injuries

TABLE V

FREQUENCY OF VARIOUS FRACTURES

METHODS OF TREATMENT AND INCIDENCE OF INFECTION

METHOD OF R.	TIBIAE FIBULAE		RADII, ULNAE		PHALANGES METACARPALS		FEMORA		HUMERI	
	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%
	14	63.6	3	13.6	1	4.5	4	18.3		
Primary suture	12	85.7	3	100.0	1	100.0	3	75.0		
Open wound	2	14.3					1	25.0		
Dakin's										
Cast	8	57.1	3	100.0	1	100.0				
Traction	8	57.1			1	100.0	2	50.0		
Amputation to control infection	1	7.1								
Traumatic amputation	1	7.1					2	50.0		
Gas gangrene	1	7.1								
Infection	4	28.6					2	50.0		

Summary: 22 open fractures, total / 6 became infected } Incidence of infection, 27.2%

Detailed analysis of the incidence of the various open fractures, their treatment as to method, and the incidence of infection occurring in the first three months of 1938. Sulfanilamide was not used, however, in the treatment of any of these fractures.

Twenty-two fractures were treated with an average elapsed time of 3 hours from the time of fracture to beginning of débridement. The average age of the group was 33 years. One death occurred at 8 hours due to a crushing injury of the chest, giving a mortality of 4.5%. This case came to autopsy.

TABLE VI

ANALYSIS OF TREATMENT AND RESULTANT INFECTION FOR FIRST THREE MONTHS OF 1938

METHOD OF R	PRIMARY SUTURE	OPEN WOUND	CAST	TRACTION
No. cases	19	3	16	19
Cases (%)	86.4	13.2	72.7	86.4
No. infections	6	1	1	4
Infections (%)	31	33.3	6.2	21.0

are frequent. Various methods of therapy have been tried without affecting this incidence. From the tables it is evident that dakinization resulting in 100 per cent infection is not the answer. Leaving the wounds open after débridement and packing with vaseline or cod liver oil ointment gauze and application of circular casts (Orr treatment) was less effective than primary suture after débridement. Immobilization in plaster with traction if necessary for maintenance of complete reduction did give better results than traction alone and was the method used most frequently.

COMPOUND FRACTURE TREATED WITH SULFANILAMIDE

The work of Colebrook and co-workers[10, 11] and of Long and Bliss[8] revealed the effective principle of prontosil to be its reduction product para-aminobenzenesulfonamide or sulfanilamide and demonstrated its in vitro bacteriostatic properties in dilutions of $1/10,000$ and higher. Subsequently, it became well known that this agent could be given subcutaneously and intraspinally in supersaturated solutions of 1 per cent without causing any local tissue damage. Furthermore in vitro studies showed its bactericidal and bacteriostatic properties to be augmented greatly in concentrations higher than could safely be obtained by systemic administration.* With these considerations in mind, one of us (N. K. J.), after complete débridement of a compound fracture of both bones of the forearm, placed 5 gm. of sulfanilamide in the wound before closure. This case was selected for our first try because of the seeming surety of infection.

The patient, a boy 12 years of age, had fallen 70 feet from a cliff into the Mississippi River. He sustained a compound fracture of both bones of the right forearm, a simple fracture of both bones of the left forearm, serious head injury, and damage to the right kidney resulting in prolonged hematuria. Three hours elapsed before the patient was brought to the hospital and, on admission, he was in profound shock. Four hours of intensive therapy, including 500 c.c. of citrated blood plus intravenous glucose, brought his blood pressure above the critical level and his body temperature from 96° to 98.6° F. Then, seven hours after injury, débridement and closure were carried out. The wound was found packed with mud driven deeply into the marrow cavities of the proximal fragments. The distal radioulnar joint and the ulnocarpal articulation were torn open and grossly contaminated. The flexor tendons were exposed and the substances of the pronator quadratus and flexor carpi

*Ranging around 1 part in 10,000.

ulnarus were badly macerated. Five gm. of sulfanilamide was placed in the wound before closure. The patient was afebrile after three days and the wound healed promptly without drainage. Due to extensive loss of substance of the radius, increased during débridement by the necessity of rongeuring away the dirty bone, approximation of the fragments was not obtained. Six weeks after surgery this gap was closed by a sliding bone graft with resultant prompt union.

Absence of suppuration in this wound with immediate healing per primum was so encouraging that all compound fractures since have been treated by introduction of crystalline sulfanilamide into the wounds after completion of meticulous débridement. These wounds are closed tightly by interrupted sutures of silk to the skin only. Closure without tension on the wound is accomplished when necessary by longitudinal splitting of the skin away from the site of the wound but parallel to the closure. Complete reduction is obtained at the time of débridement in all cases and absolute immobilization accomplished by immediate application of unpadded plaster splints held in place by either circular bandage or plaster. Reduction is maintained by continuance, when necessary, of skeletal traction in conjunction with the plaster splints.

TABLE VII

FREQUENCY OF VARIOUS FRACTURES

METHODS OF TREATMENT AND INCIDENCE OF INFECTION

METHOD OF R	TIBIAE FIBULAE		RADII ULNAE		PHALANGES METACARPALS		FEMORA		HUMERI		COMPOUND DISLOCATIONS	
	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%
	16	39.0	13	31.7	7	17.1	1	2.4	2	4.9	2	4.9
Primary suture	16	100.0	13	100.0	7	100.0	1	100.0	2	100.0	2	100.0
Open wound												
Dakin's												
Cast	16	100.0	13	100.0	7	100.0			1	50.0	1	50.0
Traction	16	100.0					1	100.0	2	100.0	1	50.0
Amputation to control infection												
Traumatic amputation												
Sulfanilamide per os	5	31.2	6	46.2	1	14.3			1	50.0	1	50.0
Sulfanilamide locally	16	100.0	13	100.0	7	100.0	1	100.0	2	100.0	2	100.0
Infection	2	12.5										

Summary: 41 open fractures, 2 became infected } Incidence of infection, 4.9%

Detailed analysis of the incidence of the various open fractures, their treatment as to method, and the incidence of infection occurring in the last nine months of 1938. Sulfanilamide was used locally in all wounds.

Forty-one fractures were treated with an average elapsed time of 2 hours and 50 minutes from the time of fracture to the beginning of débridement. The average age of the group was 32 years. Three deaths occurred, all within 72 hours, 1 from rupture of the spleen and 2 from head injuries. All came to autopsy.

Casts are not windowed and the wound is left untouched until such time as consolidation of the fracture allows replacement of splints by circular casts prior to ambulatory treatment. The minimum of 000 plain catgut suture material to accomplish hemostasis is buried in these wounds; no attempt is made to suture any deep structures except nerves.

TABLE VIII

ANALYSIS OF TREATMENT AND RESULTANT INFECTION

METHOD OF R	PRIMARY SUTURE	OPEN WOUND	CAST	TRACTION	SULFANILAMIDE PER OS	SULFANILAMIDE LOCAL
No. cases	41	--	35	17	14	41
Cases (%)	100.0	--	85.4	41.5	34.1	100
No. infections	2	--	2	2	1	2
Infections (%)	4.9	--	5.5	11.8	7.1	4.9

To date, thirty-nine compound fractures and two compound dislocations have been treated by this method (Tables VII and VIII). In two cases, both tibiae, one eight days and the other twelve days after reduction, the fractures were recompounded and subsequently became infected. The other thirty-seven healed by primary intention without local or systemic evidence of wound infection. Including the two cases in which the fractures were recompounded, the incidence of infection is 4.88 per cent. We do not feel, however, that these two cases represent primary wound infection. Briefly their histories are:

H. S., 39-year-old white male, was admitted to the Minneapolis General Hospital July 5, 1938, in shock and drunk, one hour after being struck by an automobile. He had sustained compound fractures of both tibiae and of both bones of the right forearm. Intensive treatment of the shock, including 1,000 c.c. of citrated blood, allowed débridement and reduction to be undertaken two hours after admission. Seven and one-half grams of sulfanilamide were placed in each tibial wound and 5 gm. in the forearm. The patient was a known chronic alcoholic and had been on a prolonged spree. Eight hours after admission he developed delirium tremens. Heavy sedation with paraldehyde and chloral kept him fairly quiet, however, and by the seventh day he was afebrile. The Stimson splints were windowed* over the wounds on the fifth day and the wounds were healing per primum. On the seventh day, due to general improvement, sedation was discontinued but, on the evening of the eighth day, the patient suddenly became maniacal and drove the proximal fragment of the right tibia through the healing skin wound at the site of the window in the splint. Severe infection followed with osteomyelitis due to *Staphylococcus aureus*. His other wounds healed without infection.

G. E., aged 65 years, sustained a compound "bumper" fracture of the right tibia on Nov. 4, 1938. Routine débridement with introduction of 10 gm. of crystalline sulfanilamide into the wound and primary closure were carried out three hours after injury. On the third day the patient was afebrile and remained so. However, x-rays three weeks later showed sharp anterior angulation at the fracture site due to insufficient traction. On the twenty-eighth day after added traction had failed to accomplish correction, the Stimson splints were removed to allow manipulative correction. The proximal fragment was found to have eroded its way through the

*A subsequently regretted exception to our practice.

wound and was surrounded on the sides by healthy granulations. The ventral surface was clean and viable and there was no evidence of suppuration. Sterile vaseline gauze dressings were applied and the position corrected with immobilization in circular plaster. Four weeks later the odor was characteristic of osteomyelitis and change of the cast at this time revealed purulent drainage.*

Both of these cases illustrate poor management of the fractures, not of the compound wounds. Accurate reduction must be maintained, particularly in subcutaneous bones, and windowing of casts only increases motion at the fracture site. Inspection of a healing wound is of no therapeutic value. The circumstances leading to infection in these wounds convince us that both represent reinfected wounds.

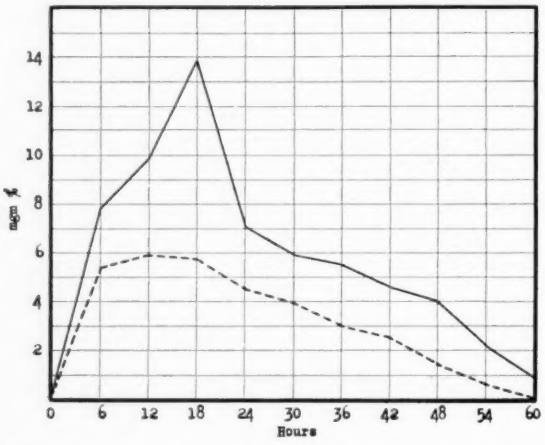

Fig. 1.—Blood concentrations of sulfanilamide in two patients after 10 gm. and 6 gm., respectively, had been buried in operative wounds.

EXPERIMENTAL

Sulfanilamide crystals buried in a wound should dissolve slowly, the drug finding its way into general circulation over a period of many hours. During this time theoretically the serum, hematoma, and local tissues of the wound are saturated with sulfanilamide at 0.8 per cent concentration, the solubility of this compound at body temperature. These considerations suggest that by placing sufficient crystalline drug in the area of contamination a concentration approaching 800 mg. per cent should be maintained for a considerable length of time. In comparison, systemic medication with sulfanilamide attempts to attain only 10 to 20 mg. per cent in the blood. Hence, by local introduction of the drug with subsequent closure of the wound, a concentration eighty times that obtained by systemic medication apparently is assured for a prolonged period in the contaminated area. This concentration is bactericidal for organisms more susceptible to sulfanilamide and, from animal experiments now in progress, appears sufficiently bacteriostatic

*One month subsequent to the submission of this paper for publication, this wound had healed without evidence of osteomyelitis, and has remained so to date (6/10/39). The corrected percentage of osteomyelitis for this series is 2.7.

TABLE IX

CONCENTRATION OF SULFANILAMIDE IN OPERATIVE WOUNDS*

AMOUNT PLACED IN WOUND	HOURS SINCE CLOSURE OF WOUND	LOCAL CONCENTRATION IN MG. %	BLOOD CONCENTRATION
5 gm.	24 hr.	666.5	2.7 mg. %
10 gm.	26 hr.	509.0	----
8 gm.	30 hr.	250.0	5.9 mg. %

*These determinations were made on filtered serum aspirated from operative wounds. Only the concentration of free sulfanilamide was determined.

even for *Staphylococcus aureus hemolyticus* to prevent infection of compound fractures.

Actual measurement of the rate of absorption and excretion of sulfanilamide in two patients in whom known amounts were placed in surgical wounds (Fig. 1) reveals that approximately sixty hours are required for the drug to disappear from the blood. It reached its highest level in the blood after approximately eighteen hours in both cases. Marshall and co-workers'[9] studies of excretion of sulfanilamide show after a single large dose by mouth that practically all the drug is eliminated in twenty-four hours. This would indicate that in the two cases studied here active absorption continued from the wound for at least thirty hours, although the rates of excretion exceeded absorption the last twelve of the thirty hours. Aspiration of serums and unorganized blood from wounds in which various amounts of the drug have been placed substantiate these deductions (Table IX). These studies are too few for definite conclusions, yet they demonstrate the validity of the theoretical foundation for the local use of sulfanilamide. Further studies now are in progress.

Experimental study of the ability of sulfanilamide to prevent infection of compound fractures in animals was carried out by fracturing the ribs of twenty-seven guinea pigs and contaminating the wound with *Staphylococcus aureus hemolyticus*. The staphylococcus culture had been obtained recently from the blood of a 13-year-old youth dying of pyemia associated with an acute osteomyelitis of the humerus. The

TABLE X

CONTROL SERIES, NO SULFANILAMIDE*

ANIMAL	RESULT
No. 1	Wound draining on 4th day
No. 2	Wound draining on 6th day
No. 3	Wound draining on 7th day
No. 11	Wound draining on 8th day
No. 9	Sacrificed on 3rd day†
No. 8	Healed on 8th day
No. 10	Healed on 9th day
Incidence of infection, 71.4%; no mortality	

*Compound fracture of rib in guinea pig. Wound heavily contaminated with *Staphylococcus aureus hemolyticus* and skin edges sutured tightly.

†Autopsy of pig showed osteomyelitis of rib, acute pleurisy, bronchopneumonia. Cultures of wound and heart's blood revealed *Staphylococcus aureus hemolyticus*.

fractures were produced by exposing the rib through the overlying muscles and cutting it with a pair of scissors. Contamination was accomplished by flooding the wound with the original blood culture of the organism. All wounds except one (No. 17) were tightly closed with skin sutures of silk. No dressings were applied and the animals then were allowed the run of their cages.

TABLE XI

EFFECT OF 0.5 GM. SULFANILAMIDE SYSTEMICALLY*

ANIMAL	RESULT
No. 13	Wound draining 4th day
No. 19	Wound draining 6th day
No. 25	Wound draining 6th day
No. 26	Wound draining 6th day
No. 27	Wound draining 4th day
No. 20	Wound draining 6th day
No. 22	Died 4th day, autopsy†
No. 23	Died 8th day, autopsy†
No. 21	Healed 8th day
No. 24	Healed 8th day
Incidence of infection, 80%; mortality, 20%	

*Compound wound identically prepared, contaminated, and closed as in control series. 0.5 gm. crystalline sulfanilamide placed subcutaneously in abdominal wall.

†Both pigs showed acute pleurisy, bronchopneumonia, and severe wound infection. Blood and wound culture revealed *Staphylococcus aureus hemolyticus.*

Consideration of the results reported here (Tables X to XII) reveals that out of seven controls five developed severe wound infection with osteomyelitis of the rib, while two healed without suppuration. In ten animals in which 0.5 gm. of sulfanilamide was placed in the wound before closure, seven healed promptly. One, sacrificed on the third day, showed no evidence of wound infection, but, on culture, *Staphylococcus aureus hemolyticus* was recovered from the wound. We feel safe, however, in assuming that this wound would not have become infected since it showed none of the edema and necrosis found in the control wounds (Nos. 9 and 17) also posted on the third day. In ten animals in which

TABLE XII

EFFECT OF 0.5 GM. SULFANILAMIDE LOCALLY*

ANIMAL	RESULT
No. 4	Healed per primum
No. 12	Healed per primum
No. 14	Healed per primum
No. 15	Healed per primum
No. 5	Healed per primum
No. 6	Healed per primum
No. 7	Healed per primum
No. 16	Sacrificed 3rd day (see autopsy)
No. 17	Infected at 8th day†
No. 18	Infected at 8th day‡
Incidence of infection, 20%; no mortality	

*Compound wound identically prepared and contaminated as in Table XI; 0.5 gm. sulfanilamide crystals placed in wound before tight closure.

†Wound left gaping, allowing drug to drain away with serum; result, infection.

‡Subjected to 3 cardiac punctures in 3 days, removing 10 c.c. blood; result, infection.

the fracture wound was produced as in the controls and contaminated with the same culture, the sulfanilamide was buried subcutaneously in a second wound on the abdomen. One, sacrificed on the third day, showed gross pus in the wound with edema and necrosis of the tissue. Seven developed osteomyelitis and two healed. From this it appears that systemically administered sulfanilamide is not effective, for infection is as frequent in this group of animals as in the control group. The two animals of the locally treated group which developed wound infections deserve mention as in one animal (No. 17) the wound was intentionally not tightly closed, but left gaping several millimeters with two loose sutures through the skin. This was done to see if it made any difference when sulfanilamide crystals, once having been placed in the wound, were allowed to escape with the serous exudate subsequently developing in such an open wound. Apparently it does. Animal 18 had cardiac punctures on the first, second, and third postoperative days to obtain blood for chemical study. This represented a loss of about 10 c.c., considerable for an animal of this size, and may have influenced the outcome. This factor, dosage, effect of particulate matter, diet, etc., and the efficacy against different strains of staphylococcus and other micro-organisms are now being studied.

These limited experimental studies demonstrate that in a wound heavily contaminated with a freshly isolated virulent resistant organism the local presence of a high concentration of sulfanilamide allows the natural local defensive factors to handle the infection in 80 per cent of the cases. In identical wounds with the same dosage of bacteria and systemically administered sulfanilamide, 80 per cent of the wounds became infected, while those receiving no sulfanilamide show 65 per cent infection.

SUMMARY

The local use of sulfanilamide has opened the way for more effective treatment of contaminated wounds. The amount to be used in each specific wound depends upon the extent of trauma and contamination. Twenty grams seems the upper limit advisable in any adult patient; less should be used in children. Five to fifteen grams have been found sufficient for average to very severe wounds.

Complete painstaking débridement aseptically carried out by frequent changes of instruments and gloves, followed by saline irrigation with primary closure of the wound without tension by interrupted sutures, still remains the fundamental treatment of contaminated wounds. Sulfanilamide will not protect against gross contamination of a badly devitalized wound containing much foreign infectious material, as it is apparently only bacteriostatic against staphylococcus, even in concentrations approaching 1 per cent. However, if the dirty and

devitalized tissue has been removed and good physiology re-established and maintained, sulfanilamide in concentrations obtainable by local introduction of the crystalline drug in sufficient amounts, by its bacteriostatic effect, will protect against infection by holding the organisms in check until local defensive forces have responded and are able to overcome the bacteria.

Good wound physiology demands immediate definitive reduction with complete immobilization maintained by traction and plaster splints where applicable. In brief, our treatment of compound fractures is thorough débridement and careful hemostasis, followed by the introduction of 5 to 15 gm. of crystalline sulfanilamide into the wound with primary closure of the skin avoiding tension. We then concentrate on treatment of the fracture. Every effort is made to accomplish complete reduction and thereafter to hold the fragments absolutely still. Excessive edema is avoided by appropriate elevation and the patient's systemic needs are met by transfusion, sedation, and intelligent medical care.

The treatment of thirty-nine compound fractures and two compound dislocations by these principles has resulted in not a single instance of primary wound infection. Two tibiae were recompounded; both became infected. Ninety-four open fractures treated by similar methods, except that sulfanilamide was not used, showed 27 per cent infection, seven cases of gas gangrene and five amputations to control infection.

Dr. A. A. Zierold, Professor of Surgery at the University of Minnesota and Chief of the Surgical Service at the Minneapolis General Hospital, showed constant interest and gave helpful suggestions in the conduct of the study. Miss Betty Hanson and Miss Cora Evenson performed the sulfanilamide determinations.

REFERENCES

1. Heister, L. A.: General System of Surgery, Ed. 8, London, 1768.
2. Ritter, H. H.: Management of Compound Fractures, Arch. Surg. **34**: 527-534, 1937.
3. Lister, Joseph: On a New Method of Treating Compound Fractures, Abscess, etc., With Observations on the Conditions of Suppuration, Lancet **1**: 326-329, 1867.
4. Carrel, A., and Dehelly, G.: The Treatment of Infected Wounds, Trans. by Herbert Child, New York, 1917, Paul B. Hoeber.
5. Böhler, Lorenz: Treatment of Fractures, English Ed. 4, 1935, William Wood & Co., pp. 499-506.
6. Foster, G. V.: Compound Fractures of the Long Bones, Surg. Gynec. & Obst. **56**: 529-538, 1933.
7. Poyner, H.: End Results in 1,000 Consecutive Industrial Fractures, Texas State J. Med. **34**: 284-288, 1938.
8. Long, Perrin H., and Bliss, Eleanor A.: Para-Amino-Benzene-Sulfonamide and Its Derivatives, J. A. M. A. **108**: 32-37, 1937.
9. Marshall, E. K., Jr., Emerson, Kendall, Jr., and Cutting, W. C.: Para-Aminobenzenesulfonamide Absorption and Excretion: Method of Determination in Urine and Blood, J. A. M. A. **108**: 953-956, 1937.
10. Colebrook, Leonard, and Kenny, Meare: Treatment of Human Puerperal Infections, and of Experimental Infections in Mice With Prontosil, Lancet **1**: 1279, 1936.
11. Colebrook, Leonard, Buttle, G. A. H., and O'Meara, R. A. Q.: Mode of Action of p-Aminobenzenesulphonamide and Prontosil in Hemolytic Streptococcal Infections, Lancet **2**: 1323, 1936.

INTERNAL FIXATION OF TROCHANTERIC FRACTURES OF THE FEMUR

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DURING the past few years a great deal of emphasis has been placed on the so-called intracapsular fractures of the hip and this has been referred to as the unsolved fracture, while the trochanteric fractures of the femur have been relatively neglected. This is probably because these fractures tend to unite regardless of the treatment and the chief problem which they present from the standpoint of local treatment is the obtaining of union without crippling deformity, the chief deformities being coxa vara and external rotation of the distal fragment. However, it is to be noted that from the standpoint of mortality the trochanteric fractures are more serious than are the so-called intracapsular fractures of the hip; and in a recent series which we have reported from the St. Louis City Hospital the mortality of the 214 trochanteric fractures was 38 per cent, while the mortality of 166 intracapsular fractures in the same hospital was 25.9 per cent. It is thus evident that not only are they more frequent, but the mortality of the trochanteric fractures is considerably greater than is that of the intracapsular fractures, even when they are treated by the same methods (Rowlette and co-workers[1]).

Since the introduction of internal fixation of the intracapsular fractures of the hip by Smith-Petersen in 1930, the mortality of these fractures has been greatly reduced where internal fixation is used routinely. We were rather slow to use the Smith-Petersen nail, because in the beginning we believed that the method of performing an open reduction and inserting the nail through a large anterolateral incision was too formidable a procedure to be undertaken lightly in the type of patients who suffer fractures of the hip. Since learning a method by which we are able to insert the nail satisfactorily with local anesthesia and practically no surgical shock (Key[2]), we have been enthusiastic in its use.

The introduction of practical methods of internal fixation in intracapsular fractures of the hip not only has resulted in the obtaining of satisfactory union in a much higher percentage of these cases than we obtained by so-called conservative methods, but it has greatly reduced the mortality. And we believe that this is important, in spite of the fact that little has been said about the mortality in the literature on the so-called conservative treatment of fractures of the hip. The chief concern has been whether or not the patients obtained bony union, and to

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judge from the literature it almost seems as though a fracture of the hip has a distinct rejuvenating effect on an aged individual.

In observing the postoperative course of patients in whom a fracture through the neck of the femur has been satisfactorily nailed or fixed by pins or a lag screw, one is impressed by the facts that they are relatively free from pain, move about in bed with relative ease, can be gotten out of bed within a few days if necessary, spend a relatively short time in the hospital, are not troubled with weakness of the legs and stiffness of the joints after their hip has united, and can resume their usual activities several months sooner than can the smaller percentage of similar patients who have obtained union under the so-called conservative method. I reiterate the term "so-called conservative treatment" because I believe that the truly conservative method of treating a fracture through the neck of the femur is to operate upon it and fix the fragments firmly together by whatever method the surgeon prefers, always providing that a surgeon capable of performing the operation skillfully and accurately is available, because the operation is not easy and requires special skill and training.

In trying to explain the high mortality of trochanteric fractures, we first have considered the fact that the trochanteric fractures occur in patients who are slightly older than are a similar group of patients who suffer fractures through the neck of the femur. The average age of the 214 patients in our series of trochanteric fractures was 66.6 years, while the average age of the 166 patients who suffered fractures through the neck of the femur was 63.8 years. That age is an important factor in the mortality of each group is indicated by the facts that the average age of the patients with trochanteric fractures who died was 73.8 years, while the average age of those who survived was 60 years; and the average age of the patients with fractures through the neck of the femur who died was 71.2 years and the average age of those who lived was 60.9 years.

It is also to be noted that the patient with a trochanteric fracture has usually suffered a more severe initial injury than has the patient with a fracture through the neck of the femur. In a fracture through the neck of the femur the head is usually twisted off of the neck with relatively little violence and the fragments are not comminuted or markedly displaced and there is little hemorrhage or damage to the soft tissues. In a trochanteric fracture the bone is broken by force transmitted up the extremity or by direct violence, as in a fall on the trochanter, and the fragments are usually comminuted with considerable displacement and hemorrhage and damage to the soft tissues.

In view of the above it is reasonable to believe that the patients with trochanteric fractures suffer more pain than do those with fractures through the neck of the femur, and clinical observations lead me to believe that they do. I also believe that one of the principal debilitating

factors and causes of death in these old people with fractures of the hip is pain. Another important factor in the mortality is immobilization of the patient, either by plaster-of-Paris casts or by traction.

The Smith-Petersen nail and its substitutes have saved many lives because they have relieved the patients of pain and have freed them from casts and splints. Even if the results were no better than by other methods, nailing would still be the humane way of treating fractures of the hip.

We have tried various methods of treating the trochanteric fractures, namely, walking plaster-of-Paris casts, the well-leg traction splints, abduction plasters, abduction plasters with traction, and the Hodgen's splint. Of the various methods at our disposal it has seemed to us that the Hodgen's splint, when properly used, made the patients more comfortable, permitted them more freedom in moving around in bed and in sitting up in bed, and obtained a better anatomic and functional result than any method which we have tried. However, the mortality has remained about the same.

In an effort to reduce this mortality we have tried to develop a method by which these fractures could be operated upon and immobilized in a manner similar to that in which our intracapsular fractures are now either nailed or pinned. In the beginning we hoped for a method which would fix these trochanteric fractures to the same degree and afford the same freedom from casts and splints and permit the patient to be gotten out of bed soon after the operation, as is the case in the fractures through the neck of the femur which have been nailed. However, it seems evident that this is not possible, because of the tendency to comminution and of the rather wide variation in the type of fracture encountered.

It now appears that it is possible, however, to so fix most, if not all, of these trochanteric fractures that the patient will be made comfortable and that he may rest in bed or move around in bed without any external splintage, unless it be merely a small weight on the leg with Buck's extension and a small pillow under the knee. This can be done by open operation and in some fractures it can be done with the Smith-Petersen nail or, if preferred, with the Moore pins; while in others it can be done with wires, the type of internal fixation depending largely upon the shape of the bone fragments and the location of the fracture.

The chief difficulty in this operation is that one is very apt to splinter the lateral cortex if one attempts to drive the nail through it without first cutting a channel for the nail. In order to obviate this we have first reduced the fracture; then, with a 3/16-inch drill, have perforated the outer cortex in the direction in which it is desired that the nail be driven and have then cut three channels for the flanges of the Smith-Petersen nail, using a small thin osteotome. In this manner it is possible to drive the nail through the outer cortex and up into the head of the

femur. We have experimented with longer nails placed lower down on the shaft of the femur, but do not believe that they are necessary and find that they are a good deal more difficult to insert.

Viewed from the standpoint of the operative procedure, trochanteric fractures fall into three groups: (1) Fractures through the base of the neck or trochanteric region with little or no visible comminution or displacement. These require no manipulative reduction and can be nailed or pinned blindly or by whatever method the surgeon chooses and the operation can be done through the straight lateral incision. These are treated as are fractures through the neck of the femur, except that the novocain is placed around the fracture and not in the hip joint and a channel is cut through the lateral cortex for the nail and the fragments are impacted very gently. The final position of the nail is checked by anteroposterior and lateral x-rays taken before the wound is closed and developed while the wound is being sutured (Key[2]). (Fig. 1.)

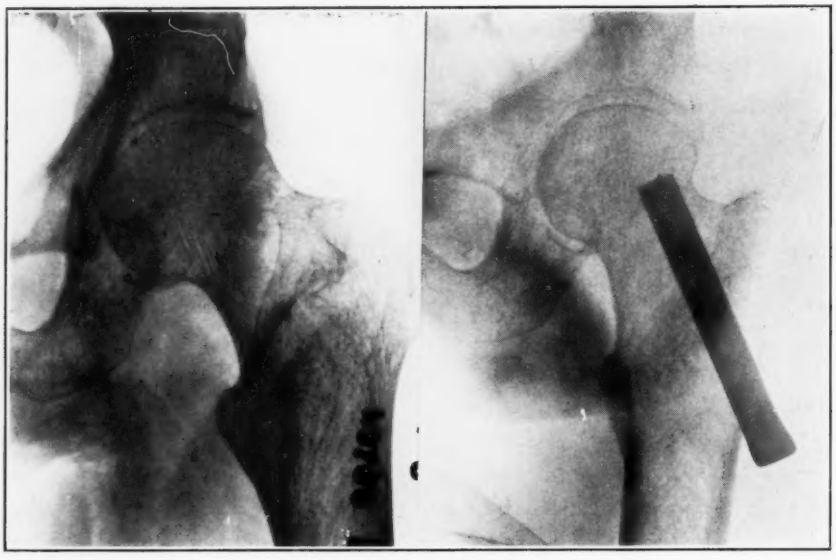

Fig. 1.—Trochanteric fracture with very slight displacement, before and after nailing. No reduction necessary. Nail should extend farther into the head, but in this instance there were no complications.

2. The usual trochanteric fracture with considerable separation and comminution, but with the lateral cortex of the shaft of the femur intact up to or beyond a point about in line with the superior surface of the neck of the femur. After manipulative reduction these are nailed through a lateral incision or by direct exposure through an oblique incision as is described below. (Figs. 2 to 7.)

3. Fractures involving the lateral cortex of the femur below the level of the line through the superior surface of the neck of the femur. These are really subtrochanteric fractures and cannot be nailed, but can be fixed with wires or a Parham band so that the fragments are fairly stable. (Fig. 7.)

OPERATIVE TECHNIQUE

Unless the patient is in a condition of shock, the operation is performed as soon after the patient is admitted to the hospital as possible. Before the patient is removed from bed, 20 to 30 c.c. of 1 per cent solution of novocain is injected into the hematoma at the site of the fracture. This can be done by directing the needle downward and inward on the anterolateral surface of the thigh at the level of the fracture and aiming at the line of the fracture as determined by the x-ray. When the needle is in the hematoma, blood can be drawn back into the syringe. Then the patient is placed on an ordinary operating table with a tunnel under him to facilitate placing the carrier for the x-ray films under the hip. The entire limb is painted with tincture of mer-

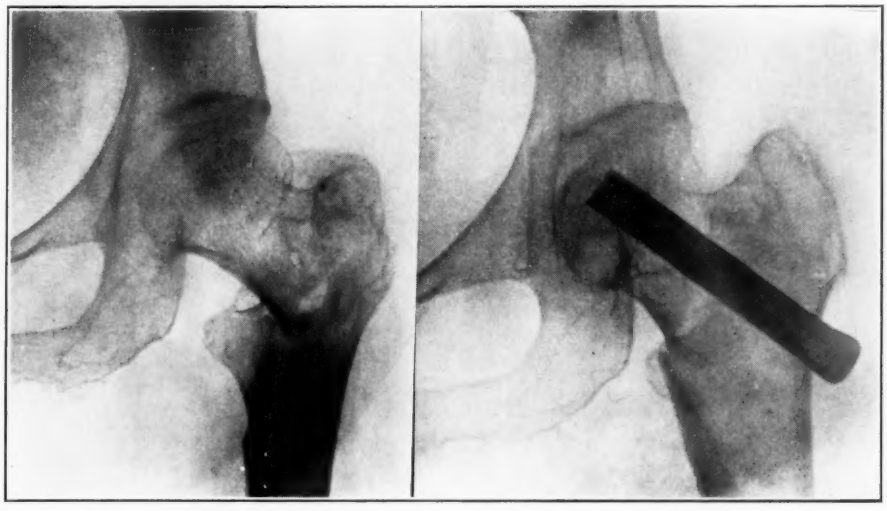

Fig. 2.—Trochanteric fracture before and after satisfactory nailing.

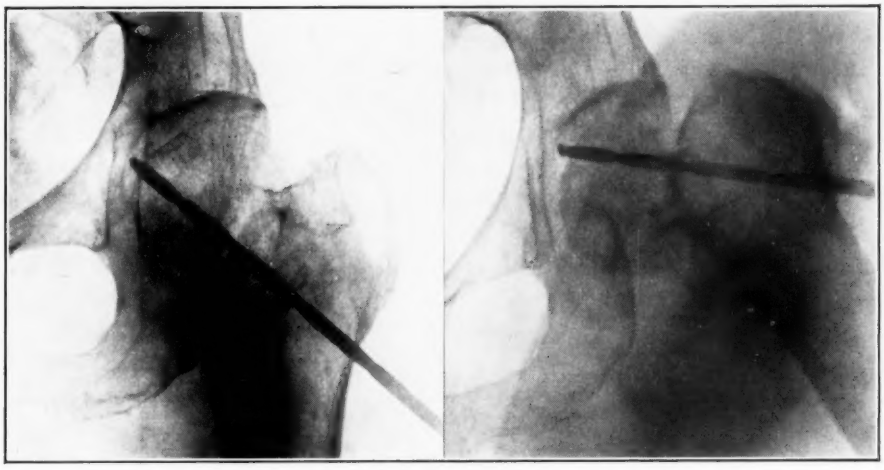

Fig. 3.—Same as Fig. 2, anteroposterior and lateral views, showing the guiding drill in position.

thiolate or iodine and the limb so draped that it can be manipulated by the operating team.

The skin is infiltrated in the line of the incision with 1 per cent novocain which contains three drops of adrenalin to the ounce. Then the deeper tissues are infiltrated with 0.5 per cent novocain which also contains three drops of adrenalin to the ounce. After the skin and deeper tissues have been infiltrated with novocain, the extremity is rotated inward to about 30 degrees beyond the midposition. If the fragments are displaced, strong manual traction is made on the extremity and then it is rotated inward about 30 degrees and abducted about 30 degrees. The limb is maintained in this position by an assistant until the nail has been driven home into the head of the femur. When the lateral x-rays are being taken, the internal rotation and abduction are maintained while the hip is carefully flexed to about 90 degrees.

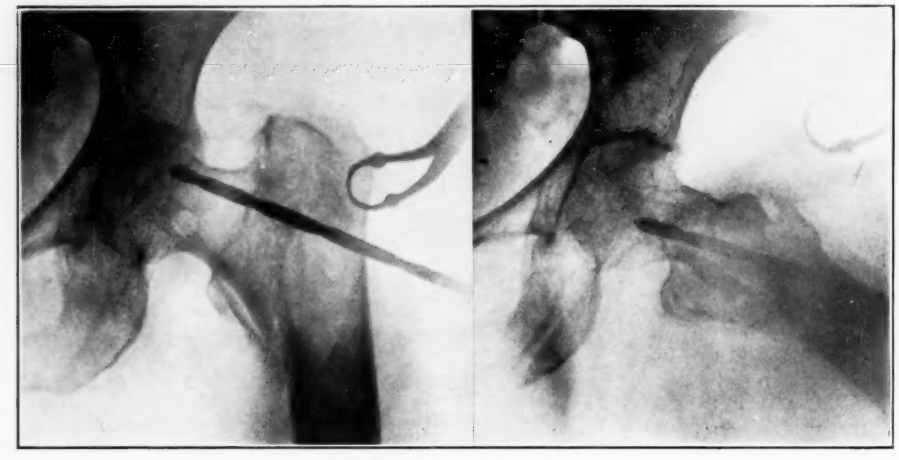

Fig. 4.—Trochanteric fracture incompletely reduced, with guiding drill in position. Drill is too high.

The straight lateral incision begins on the lateral surface of the thigh opposite the tip of the trochanter and extends downward for 5 or 6 inches. It is carried down through the deep fascia to expose the lateral surface of the trochanter and the vastus lateralis muscle. This muscle is split in its upper 2 inches and its origin cut from the side of the trochanter to expose the lateral surface of the femur below the trochanter.

The anterior surface of the trochanteric region and the base of the neck now can be palpated and it is possible to determine roughly the accuracy of the reduction and the position of the neck and head of the femur. If the reduction is not satisfactory, the limb can be manipulated again while the surgeon's finger palpates the line of the fracture. It is to be noted that the smaller fragments and the lesser trochanter are ignored and that one attempts only to restore the two major fragments to a satisfactory functional position.

At a point as low down on the lateral surface of the shaft as is consistent with passing the nail through the neck of the femur, a 3/16-inch drill is inserted for a distance of about 3 inches. This point is about 1 inch below the base of the trochanter and the drill is directed upward and inward towards the center of the head, as determined by palpation and the preoperative x-rays. If reduction by traction has been necessary, due allowance for this is made in directing the drill. The direction of the drill in the anteroposterior plane is controlled by keeping the drill horizontal, since the long axis of the neck of the femur lies approximately in the horizontal plane when the foot is rotated inward about 30 degrees. (Fig. 4.)

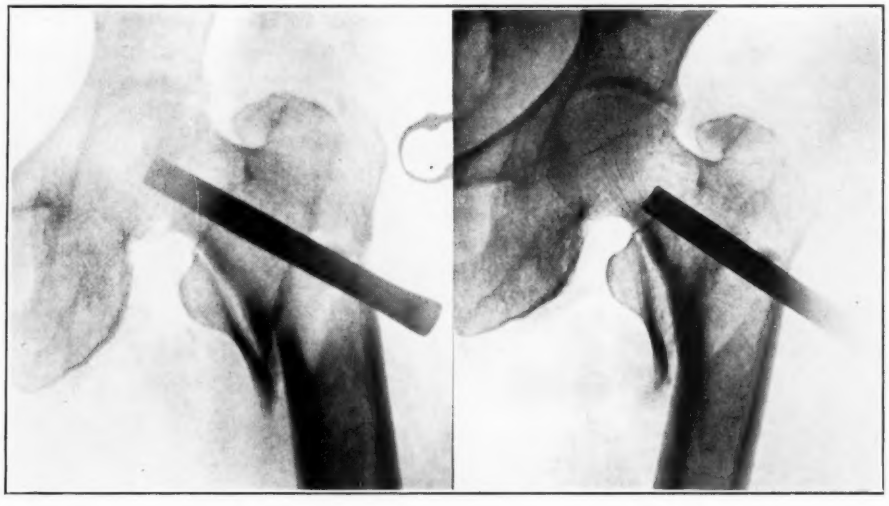

Fig. 5.—Same as Fig. 4, after nailing. The nail was shifted downward from the course of the drill, but did not extend far enough into the head and on the left the nail had slipped three weeks later.

With the drill point in place and binding the two major fragments together, anteroposterior and lateral x-rays now are made. This latter can be done by carefully flexing the hip to 90 degrees while maintaining the internal rotation and moderate abduction. While these films are being developed, the drill is removed and the exact length of the drill point which has entered the bone is noted, as this will aid in determining the length of nail to be used in this case. Now, with a small thin osteotome, three channels are cut for the flanges of the Smith-Petersen nail. This cutting of the channels must be done carefully and gently; otherwise, one may split the cortex. When completed they should permit driving the nail with relatively little resistance. After the channels have been completed, the drill is reinserted and with this size drill no difficulty is encountered in pushing it back into the hole in the head if the extremity has not been moved while it was out.

If the x-rays show the drill to be pointing in the proper direction, it is withdrawn and a Smith-Petersen nail of the proper length is driven

home into the head of the femur, directing it as nearly as possible in the line taken by the drill. If the surgeon has difficulty in keeping this line in mind while the nail is being substituted for the drill, a small drill can be placed in the shaft about ½ inch from and parallel to the drill which is directed towards the head of the femur, and this can then be used as a guide in directing the nail. The length of nail desired is determined by adding the distance of the tip of the drill from the articular surface of the head, as seen in the x-rays, to the length of the drill which penetrated the bone.

If the position of the drill is not satisfactory, then the nail is started in the drill hole with the three channels for the flanges of the nail cut around it and is not driven in the direction taken by the drill, but is tilted up or down or forward or backward as much as necessary to direct it towards the center of the head of the femur and is then driven home. The fragments are then impacted very gently and anteroposterior and lateral x-rays are made. The wound is closed without drainage and if the position of the nail is found to be satisfactory the patient is placed in bed with a small pillow under the knee and with the leg in a position of slight abduction.

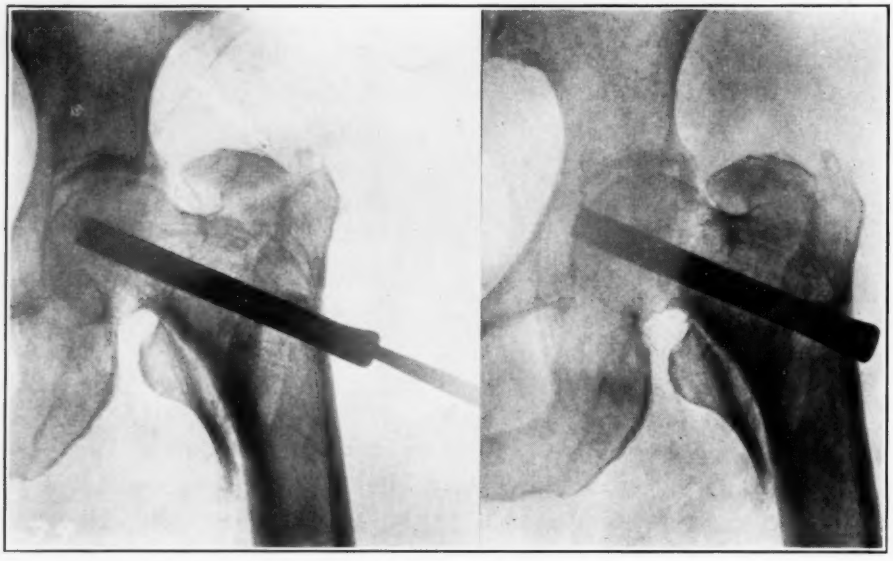

Fig. 6.—Same as Fig. 5, showing nail being driven back into the head with a pin and on the right the nail is driven far into the head of the femur.

If the final x-rays show that the nail is not fixed firmly in the head of the femur, the wound is opened immediately and the nail is withdrawn and driven in again in the corrected direction.

If the surgeon prefers, a cannulated nail driven over a Kirschner wire guide may be used, or he may use one of the several instruments now on the market which are supposed to aid in placing the nail properly. Likewise, he may expose the fracture through an oblique

incision and drive the nail under direct vision in a manner similar to that used by Cubbins, Callahan, and Scuderi.[3] We prefer this method when we are using a general anesthetic, as the guiding drill and preliminary x-rays are not necessary and consequently the time consumed in the operation is shorter.

In the subtrochanteric fractures it is not possible to use a nail, but in old, debilitated patients the fracture can be exposed and reduced under local anesthesia and the fragments so fixed with wires that good position can be maintained with a slight amount of traction or in a single low spica cast (Fig. 7).

We have not used the Moore pins because we routinely use the Smith-Petersen nail. However, I believe that the pins will work equally well, and they have the advantage that they can be drilled through the lateral cortex without danger of splitting it. In our series the cutting of the channels for the flanges of the nail has been a satisfactory solution and we have not split the lateral cortex. But we are careful not to try to sink the head of a nail with tapering flanges deeply into the cortex.

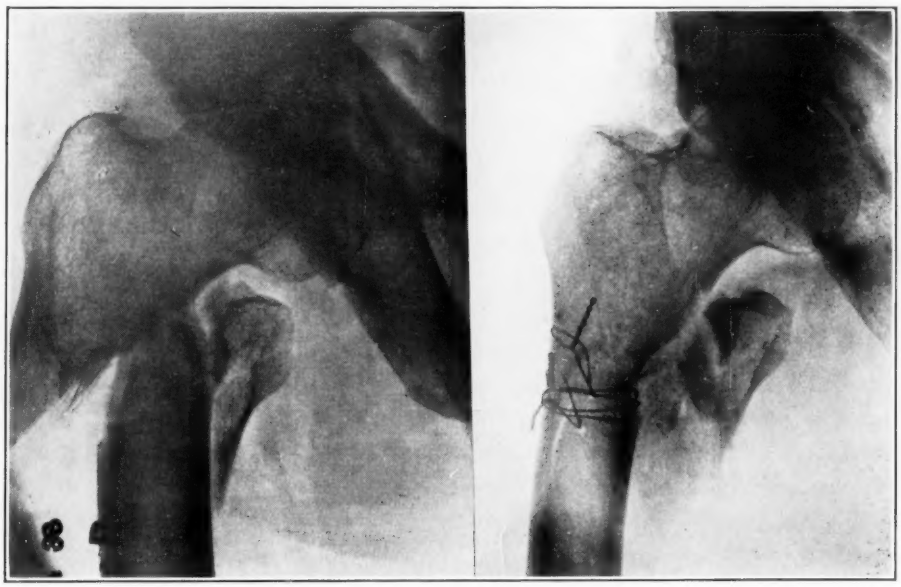

Fig. 7.—Subtrochanteric fracture before and after open reduction and fixation with wire. Nailing not possible in this type.

POSTOPERATIVE CARE

The postoperative care of patients in whom a trochanteric fracture has been nailed is usually more difficult than is that of an intracapsular fracture which has been subjected to the same procedure. In the trochanteric fracture the fragments are not so firmly bound together and we maintain the extremity in abduction with a pillow under the knee. The patient may sit up at will, but the leg must be lifted and supported on pillows when the patient is turned on the sound side. At the end

of from two to four weeks the patient may sit on the edge of the bed and dangle the feet or even be helped into a chair, but weight-bearing and even walking on crutches without weight-bearing are discouraged until union is quite firm. Very heavy patients do better in a fracture bed, but the average patient prefers an ordinary hospital bed.

When properly inserted, the nail stabilizes the hip sufficiently to prevent outward rotation of the distal fragment and also sufficiently to prevent it from being displaced forward or backward or inward or outward. And usually it prevents the development of a coxa vara. However, in comminuted trochanteric fractures x-rays should be taken about two weeks after nailing and if there is any tendency to a slipping up of the distal fragment (coxa vara), traction (10 to 15 pounds) should be applied to the extremity until the deformity is corrected and the fracture is fairly well healed.

The period during which these patients must remain in bed will depend largely upon the type of fracture and upon the adequacy of the immobilization. If adequate immobilization can be secured, as was done in the case of the patient shown in Fig. 1, the patient may be permitted out of bed on crutches at the end of two weeks. On the other hand, in fractures such as that illustrated in Fig. 7, the period of rest in bed must be prolonged to from eight to twelve weeks or whenever union appears sufficiently solid. In this patient a plaster spica cast was applied to the hip two weeks after the operation and he was sent home to convalesce.

DIFFICULTIES

In nailing a series of fifteen trochanteric fractures we have had no operative infection and no immediate mortality, but two of the patients died about six months after the injury. Each of these patients was over 80 years of age and apparently the fracture of the hip had healed and was not definitely related to the terminal illness. We believe that as the series is increased the mortality will be about 10 per cent.

In internally fixing trochanteric fractures we have encountered certain difficulties which we believe can be avoided by more care, experience, and skill on the part of the surgeon. In my first case the nail was driven through the head of the femur and into the floor of the acetabulum. This can be avoided if it is remembered that in a comminuted trochanteric fracture the base of the neck may be telescoped into the trochanteric region and the distance from the lateral cortex to the head of the femur may be ½ inch or more less than it is in the normal hip. In nailing a fracture of this type a relatively short nail should be used (Fig. 2).

A more frequent mistake is not to reduce adequately the fracture before the nail is inserted or to fail to maintain reduction. After displacement has occurred, these fractures cannot be reduced and locked by

internal rotation and abduction as can an intracapsular fracture. Consequently, unless care is taken to prevent it the fracture may be nailed with a varus deformity. (Figs. 4 and 5.)

A third mistake which we have made is not driving the nail deeply enough into the firm cancellous bone of the head. In one instance a nail backed out during the fourth week after the operation. It was driven back in place with a pin inserted through a small incision under local anesthesia (Figs. 5 and 6).

It is to be emphasized that the nailing of a trochanteric fracture in which the fragments have been displaced requires more care and skill on the part of the surgeon than does the reduction and nailing of an intracapsular fracture. However, it is possible to nail practically all of these fractures under local anesthesia and with practically no shock to the patient and we believe that if these fractures are nailed properly there will result a very definite decrease in the mortality which, as has been noted above, is higher than is the mortality of the intracapsular fractures. It is also to be emphasized that these fractures cannot be fixed as firmly as the intracapsular fractures and that postoperatively the patients must remain in bed longer and require more care than do patients in whom intracapsular fractures have been nailed successfully.

CONCLUSIONS

1. Trochanteric fractures occur more frequently than do intracapsular fractures of the hip.

2. The mortality is greater in trochanteric fractures than it is in intracapsular fractures of the hip.

3. It is believed that internal fixation of trochanteric fractures will appreciably reduce the mortality and add much to the comfort of the patients.

4. Methods of internally fixing trochanteric fractures are described.

REFERENCES

1. Rowlette, A., Haslem, J. R., Siegert, R. D., Morris, H. D., and Key, J. A.: Results Obtained by Subcutaneous Pinning of Fractures Through the Neck of the Femur, J. A. M. A. **107**: 1610, 1936.
2. Key, J. A.: Insertion of Smith-Petersen Nail for Intracapsular Fractures of the Femur, Am. J. Surg. **36**: 466, 1937.
3. Cubbins, W. R., Callahan, J. J., and Scuderi, C. S.: Fractures of the Neck of the Femur, Surg., Gynec. & Obst. **68**: 87, 1939.

DISCOID CARTILAGE, TRIGGER KNEE

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AN ARTICLE by Middleton[1] in the October, 1936, issue of the *British Journal of Surgery* gives the bibliography and the historical events in cases of discoid cartilage from 1889 to the present time. There is a reference to the classical study of Jaroschy, of Prague, and the information is given that forty-nine cases of this condition have been reported to date.

It has been well recognized by several writers that discoid cartilage represents a persistence of the embryonic external semilunar cartilage. There is also a reference to Fisher's article, in which he considers the relation of trigger knee to cystic degeneration of the external semilunar cartilage.

There have been three recognized cases in the past few years at the Boston Children's Hospital and one adult private case of mine. These cases are reported in detail:

CASE 1.—E. B. (A147,162), female, aged 4½ years. Admitted, Oct. 23, 1933; discharged, Nov. 16, 1933.

History.—Admitted for pain in the left knee of two months' duration. Child's mother gave a history of snapping sensation in the knee when it was straightened out. No other complaints and no history of injury.

Physical Examination.—General physical examination negative. Local examination showed a clicking sound in the left knee which occurred during extension. Something could be felt to snap when the knee was in about 107° of extension. The difficulty appeared to be in the region of the external lateral ligament in the joint line. Laboratory reports were all negative.

Operation.—Oct. 25, 1933, under general anesthesia, tourniquet was applied and the knee was exposed through a Kocher incision. The incision was carried down to the capsule. When the knee was extended from the flexed position, a large mass could be seen coming into prominence during extension and a snap could be heard. Opening the joint, the large mass was found to be a discoid meniscus about three times as thick and wide as normal. The cartilage disk was completely excised. Wound closed with silk. Physiotherapy was given to develop the quadriceps. On Sept. 19, 1935, the knee was well.

CASE 2.—M. T. (A178,328), female, aged 6 years, 7 months. Admitted, Jan. 29, 1934; discharged, March 8, 1934.

History.—There was a history of frequent attacks of pain in the left knee with swelling and tenderness on the lateral aspect of three months' duration. There was increased pain on motion and inability to extend the knee beyond 135°. There was no clicking and no history of trauma. The attacks occurred once to three

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times weekly and were of two or three days' duration. The attacks were sudden in onset and occurred while sitting and during active play.

Physical Examination.—Positive findings, confined to the left knee, showed ¾ inch atrophy of the thigh and ½ inch atrophy of the calf. There was a definite mass over the head of the lateral condyle of the tibia. This mass could be pushed back into the joint on complete extension of the knee. Laboratory findings were negative.

Operation.—Under general anesthesia a tourniquet was applied and a Kocher incision made on the lateral aspect and carried down to the capsule. A definite mass could be felt on examination and this seemed to project over the joint. The joint was entered and the external meniscus was found to be thickened over its posterolateral border and in front of the external lateral ligament. It covered practically the whole articular surface of the outer tuberosity of the tibia. When this was dissected free, the knee could be completely extended without difficulty.

Postoperative Note.—There was mild effusion of the joint. Active motion was started on the fifth day. Wound healed by primary intention. Six months later there was normal motion in the knee without pain or recurrence of the previous symptoms.

CASE 3.—B. C. (A181,732), female, aged 2 years, 8 months. Admitted, March 22, 1937; discharged, April 13, 1937.

History.—Mother stated the child had always walked rather peculiarly, throwing one knee in and walking with flatfeet. The summer before, she was struck on left knee by her uncle while playing. The pain occurred only at that time, but the next day the knee was slightly swollen. She was taken to local doctor who found nothing unusual. X-rays were taken a week later but revealed no bony deformity. After a period of five weeks, during which time no improvement was noted, patient was referred to the outpatient department. When first seen there, the only clinical findings were those of slight limitation in extension and slight fullness on the lateral aspect of the left knee. X-rays then revealed no bony anomaly. The patient complained of pain when the knee was extended past the point of limitation. Child had continued to be active in all exercises. Child stated that one morning left knee snapped and after this she was able to extend the knee more than before and there was less swelling.

Physical Examination.—When the patient stood, there was definite genu valgum with both feet pronated, the left more than the right. Stood with left knee flexed and right knee in hyperextension. Very slight evidence of limp on walking. Examination of left knee showed no increased fluid. Palpable and observable fullness over femoral tibial fossa on the lateral aspect. The capsule in this area was slightly thickened. No movable or definite palpable mass was found, although joint line was not easily discernible. Tenderness over the tibial condyle laterally, just below the articular margin about 2 inches posterior to the lateral border of the patella and located about midway along the area of increased fullness. No evidence of any loose bodies. No locking. Lateral mobility slightly less than on the right. Rotation mobility about the same. Anterior and posterior mobility slightly decreased.

Operation.—Under general anesthesia, curved lateral incision was made. Knee joint opened and no increased fluid found. Lateral cartilage markedly enlarged and thickened and ran medially across the external tuberosity of the tibia, nearly covering the articular surface, and was attached at the medial aspect to the origin of the posterior limb of the cartilage. Narrow isthmus of synovial membrane connecting the anterior and posterior portions of the cartilage. Whole external semilunar cartilage removed. Wound closed.

Postoperative Note.—Good postoperative recovery. Stitches removed on the tenth day. Physiotherapy started on the thirteenth day.

Pathological Examination.—Examination of specimen removed at operation showed it to consist of fibrocartilage, highly collagenous tissue of the tendinous type and a moderate amount of fat. There was one *large cystic* area measuring 2 by 3 mm. in diameter in the junction between the fibrocartilage and tendon which was filled with pale blue-staining amorphous, mucoid-like material. The margins of the cyst were composed of flattened connective tissue cells and occasional foci of acute edema and degeneration of connective tissue. There were several small areas of similar cystic degeneration in the surrounding connective tissue and fibrocartilage. One of these was of special interest in that it apparently represented a very early lesion. This represented a small central cystic space containing a few macrophage-like cells surrounded by a fibrillar degenerating connective tissue in which there were a few young fibroblasts. *Diagnosis:* Cyst of the external semilunar cartilage with degeneration.

Ten months later, left knee showed complete extension and complete flexion. No fluid present. No instability. Quadriceps showed excellent tone and patient had a normal walking and running gait.

Fig. 1.—Joint opened, exposing cartilage.

Case 4.—E. R., female, aged 31 years.

History.—At age of 4 or 5 years, she limped and was treated then with baking and massage. Had no particular discomfort at that time and noticed no pain about hips but became tired after walking any distance. Was unable to cross knees or get down from a high step without jumping. In 1934, three years previously, she was trying to put on overshoes and felt something give away on the inner aspect of the left knee. She was unable to straighten knee for several hours. Knee was treated with adhesive strapping. Had no trouble until January, 1937, when she had recurrence and knee became swollen. Treated with bandage but had another recurrence of original trouble three days later. This time it was on the outer side of the knee. Patient had three similar attacks. She was first seen by me in February, 1937. For two weeks she had been unable to get her knee perfectly straight.

Physical Examination.—The left knee lacked about 5° of complete extension. There was full range of motion in flexion. Slight excess of fluid in the joint. Definite marked local tenderness over the external semilunar cartilage. No tenderness elsewhere over knee. Forced internal rotation of the foot produced discomfort

Fig. 2.—Cartilage being dissected from the joint.

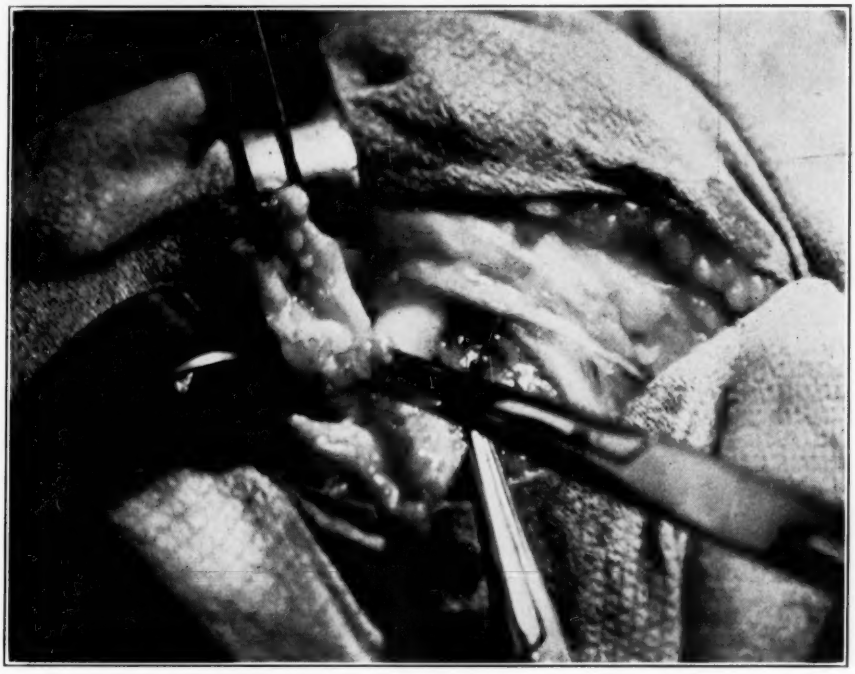

Fig. 3.—Cartilage practically cleared from the joint.

in the region of the external semilunar cartilage. Walked with a mild limp favoring the left knee. Walked with slight positive Trendelenburg. The hips were examined and showed some limitation of motion. The x-rays showed marked deformity of the heads of both femora suggestive of an old bilateral coxa plana. The x-rays of the knees were essentially negative, except for lipping as a result of hypertrophic arthritis.

Operation.—Under general anesthesia, curved Kocher incision was made over the lateral aspect. Capsule opened. Cystic mass was opened into superficially to the cartilage and the inner surface was finely granular. Synovial membrane was dissected free from the femur and tibia. Spur formation was plentiful and the reaction about the synovial membrane was consistent with hypertrophic arthritis and synovitis. The cartilage seemed larger than normal. The external lateral ligament was cut in order to expose the cartilage externally. The cartilage, which was a discoid affair, was removed. The articular cartilage over the lateral condyle was eroded over an area of about 3 cm. in diameter, exposing bare bone. The capsule was brought together and closed with fine silk; the iliotibial band was reunited and the subcutaneous fat and skin closed with interrupted sutures. Compression bandage and plaster splint applied.

Fig. 4.—Discoid cartilage. Irregularly shaped cartilage removed in Case 3. Disk not completed by cartilaginous tissue but by membranous tissue which does not show in specimen.

Postoperative Note.—Half of the sutures removed on eighth postoperative day and the rest on the tenth. Quadriceps settings started.

Pathologic Examination.—Cystic degeneration of the cartilage and discoid cartilage.

The patient was last seen in May, 1937, about three months after operation, and at that time had no fluid in knee. Could flex it to at least 10° or 15° beyond a right angle. Extension normal. Good gait.

Internal derangement of the knee in children, due to injuries of the semilunar cartilage, is extremely rare. Lesions of the external semilunar cartilage in adults are relatively rare. Lesions of the external semilunar cartilage in children are much more common than those of the internal semilunar cartilage. These lesions in children cannot be explained on a hypothesis of trauma. They can be explained, however, on an embryologic basis. The discoid cartilage lesion is due to a persistence of the embryonic form of the cartilage which is disk shaped and in most instances persists as a disk after birth. In some cases the cartilage is not in the form of a disk but is more or less shaped as shown in Fig. 4, with a membranous connection filling in the defect so that it resembles an incomplete disk. As growth goes on, these cartilages are prone to become thickened in their anterior, lateral, or posterior parts so that there is not enough room for them in the knee joint. As a result of this thickening, they are gradually pushed out of the joint in different positions of the knee if the thickened portion of the cartilage gets caught between the articular surfaces.

Fig. 5.—Shows tibial surface and cystic degeneration of specimen shown in Fig. 4.

In the adult and child cystic degeneration may take place in a portion of the cartilage.

Symptoms.—In some instances symptoms of trigger knee may be noticed by the parents, but the symptoms and signs are more apparent after weight-bearing has begun and the muscles and ligaments have tightened physiologically.

These children complain of attacks of sudden pain in the knee which may appear at any time when the cartilage catches on bending or extending the knee. There is usually a limp present, and there may be swelling of the knee.

Examination.—Examination shows atrophy of the calf and thigh muscles, and fluid may be present in the joint. The joint may be locked in flexion or rarely in extension. On palpating the extended knee, a small tumor-like mass is felt over the lateral aspect of the joint in the region of the lateral ligament. This mass usually disappears on flexion and reappears on extension with a definite snap and a jerking sensation. If it is large, the snap may occur both on flexion and extension.

Discoid Cartilage.—Discoid cartilage is a persistent embryonic cartilage which varies in shape and thickness. It may completely cover the external articular surface of the head of the tibia or it may cover only a part of the surface. In one case there was a thin membrane covering that portion of the articular surfaces not covered by the discoid cartilage. If the cartilage is thicker than the normal external semilunar, there will be symptoms of an internal derangement of the knee. It is possible that a discoid cartilage may be present without symptoms.

Treatment.—The treatment for discoid cartilage giving rise to symptoms is complete extirpation of the cartilage as soon as the diagnosis is made. In cases complicated by cysts of the cartilage, the cartilage must be removed. If this is not done, the mechanical irritation of a cartilage, which is becoming enlarged from the cyst formation, is likely to injure the articular surfaces and produce arthritic changes.

REFERENCE

1. Middleton, D. Stuart: Congenital Disc-Shaped Lateral Meniscus With Snapping Knee, Brit. J. Surg. **24**: 246-254, 1936.

PANTALAR ARTHRODESIS IN POLIOMYELITIS*

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BY THE TERM pantalar arthrodesis is meant the surgical fusion of the tibiotalar, talonavicular, talocalcaneal, and calcaneocuboid articulations; in other words, the surgical arthrodesis of the ankle and subtalar joints.

The purpose of this paper is to give briefly the indications, the technique, and the results of the operation as performed for anterior poliomyelitis.

The procedure was originally carried out only in cases of tuberculosis and the first pantalar arthrodesis performed at the New York Orthopaedic Dispensary and Hospital was in 1920, for tuberculosis involving the ankle and subtalar joints. Additional cases were operated upon in the succeeding years and, after the observation that such an extensive arthrodesis did not materially impair the patient's gait, the procedure was deemed applicable to other conditions.

In 1931 the operation was performed for deformity and instability of the foot due to poliomyelitis, and by Jan. 1, 1937, eighty-two such patients had been operated upon.

The reason for doing pantalar arthrodeses in poliomyelitis is to stabilize the foot in good lateral position and to improve an awkward gait in patients who have the deformities of calcaneus or equinus, combined with lateral instability of the foot. An additional reason, in cases with similar deformities, is the stabilization of the knee when that joint has been made unstable by paralysis of the quadriceps muscles. Braces which have been worn for instability often may be discarded following operation, and walking becomes easier, smoother, and less fatiguing.

It is felt that the operation should be done in two stages. Five patients did have the combined operation performed in one stage, but this is not recommended, because of the length of operating time and the difficulty in obtaining correction of all deformities simultaneously.

It is considered best to do the subtalar arthrodesis before the ankle fusion. If the ankle is fused first, it is very difficult to maintain the right degree of equinus and correct the other deformities. On the other hand, if the subtalar arthrodesis is done first, then all attention can be concentrated upon putting the foot at the desired degree of equinus, the importance of which will be brought out later.

The surgical technique recommended for the subtalar arthrodesis is that described by Hoke, which is well known without further description. For fusing the ankle, the multiple chip method is recommended.

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The joint is approached by a five-inch anterior incision in the sagittal plane, dividing the skin and subcutaneous tissues and, more deeply, the transverse crural and cruciate ligaments. The extensor hallucis longus and anterior tibial tendons, together with the dorsalis pedis artery, vein, and deep peroneal nerve, are retracted medially, and the extensor digitorum longus tendons laterally. The anterior lateral malleolar artery and vein are divided and ligated. The periosteum over the tibial diaphysis is incised, but one should avoid stripping the periosteum over the epiphyseal cartilage.

The articular cartilage is then entirely removed together with bone sufficient to correct the deformity or to put the foot in the desired position. With a curved osteotome, opposing surfaces are roughened so as to make a soft bed of cancellous bone. Using the same incision, bone is gouged from the lower tibial diaphysis well above the epiphyseal cartilage and small chips are wedged into the joint, particularly between the talus and malleoli. This is important because it eliminates the space created by removing the cartilage and insures firm contact of bone surfaces. A few bone chips are placed over the anterior aspect of the joint.

The capsule, periosteum, and other deep tissues are reunited with interrupted catgut sutures and the skin with silk sutures.

These operations, of course, cannot be performed until the bones of the foot are sufficiently developed. Such development varies and must be judged by roentgenograms. Usually the patient has to be at least 8 years old before the subtalar arthrodesis can be performed. The ankle fusion can be done any time after six weeks from the date of the subtalar arthrodesis. When the distal epiphyseal cartilage of the tibia is still present, care must be taken not to injure it.

The most important single factor in pantalar arthrodesis is the position of the foot following operation. It is assumed without discussion that following a subtalar arthrodesis, the foot should not be immobilized in varus or in too much valgus.

The entire success of the subsequent ankle fusion depends upon immobilizing the foot in the proper degree of equinus. Too little equinus results in a stiff, peg-leg gait. The other extreme of too much equinus produces a halting gait, in which the foot is kept in front of the body and walking is accomplished by taking short steps, pushing the arthrodesed foot ahead, and bringing the opposite foot up from the rear. However, when the proper degree of equinus is present, the patient walks with no perceptible limp, providing there is no extraneous deformity to affect the gait.

Normal individuals were studied to note the relationship between the height of the heel of the shoe and the degree of equinus of the foot. This was done by taking lateral roentgenograms of the foot while the individual was wearing a shoe and measuring the degree of equinus of the foot and the height of the heel. The ordinary heel of a man's shoe is

about ¾ inch in height and produces an equinus of about 5°. The ordinary heel of a woman's shoe is about 2¼ inches in height and produces an equinus of the foot of about 15°. Therefore, one can estimate the degree of equinus in which a foot should be placed by determining the height of the heel the patient ordinarily wears, allowing 5° of equinus for each ¾ inch of heel height.

For these reasons, it is obvious that one must determine before operation the type of heel the patient most commonly will wear. It is difficult to state the exact degree of equinus which will be applicable to all patients. However, from experience, approximately 10° of equinus has been found to be most satisfactory, because the patients who obtained an excellent result were those with a permanent equinus ranging from 99 to 103°. Other patients with a little less or a little more equinus than this obtained good results by the simple expedient of small adjustments in the height of the heel.

It is very difficult to obtain the proper degree of equinus at the time of operation, because during application of the cast following operation estimation of equinus has to be made by the surgeon's judgment. This is extremely variable, even with the same surgeon. It is absolutely necessary, therefore, to check the position of the foot within a few days after operation with lateral roentgenograms, and whenever the equinus is too great or too little the plaster must be wedged. In this series, wedging was necessary in 21 per cent of the cases.

Estimation of equinus by roentgenograms is made by two lines, one drawn down the midshaft of the tibia and the other drawn from the base of the head of the first metatarsal bone to the base of the calcaneum. The angle at the point subtended by these two lines is the degree of equinus. Errors in determining the actual equinus of the foot will be made if the amount of anteroposterior motion at the anterior tarsal joints is not taken into account. For example, let us assume that there are 10° of anteroposterior forefoot motion extending from 100° dorsiflexion to 90° dorsiflexion. Should the forefoot be drawn down, when the cast is applied after operation, the x-ray measurement would reveal the foot to be at 100°. But when the patient stands on the foot without plaster, the forefoot will move upward 10°, placing the foot in an actual position of only 90° dorsiflexion, which is insufficient for a good gait. This very thing occurred in 6 per cent of the patients in this series.

Therefore, in order to obtain an accurate roentgenographic reading of equinus, the entire foot first must be placed in marked equinus and then the forefoot must be brought up until the desired degree of equinus is obtained.

About 35 per cent of the patients required some sort of heel or sole lifts. These varied between raising the heel ¼ inch in height to promote a smoother gait and raising the heel 3 inches and the sole 2 inches to compensate for 3 inches shortening of the leg.

Obviously, increasing the thickness of the sole or heel materially aids a limp that is due to a short extremity, but it has no effect on a limp or lurch due to poor or absent abductor muscles.

Heel lifts also are employed to give longer strides, to allow the heel to touch the floor, and to improve the gait. This does not hold, however, in cases of ankle fusion to stabilize the knee, because here too high a heel may cause the knee to give way. Whenever sufficient equinus has not been obtained to stabilize the knee, it is best to employ an anterior heel; that is, a thick metatarsal bar across the sole of the shoe. Thus, when the foot is bearing weight, the tibia is held posteriorly, forcing the knee into extension.

When stability of the knee is not a problem and one has obtained too much equinus, the heel may be raised accordingly. Such building-up of the heel will work very well for as much as 20° equinus, provided good forefoot motion is present.

Revision of either the ankle fusion or the subtalar arthrodesis may be necessary for pseudarthrosis, varus, valgus, and improper equinus. Eleven of the eighty-two patients (13 per cent) required secondary operations. Five revisions were necessary for pseudarthrosis, three of which occurred at the ankle joint and two of which occurred at one or more of the subtalar joints. Two subtalar arthrodeses were revised because of varus. In one instance it was necessary to revise a subtalar arthrodesis for excessive valgus. Three revisions were done, two at the ankle and one at the subtalar joints, for improper equinus.

The results of pantalar arthrodesis in the eighty-two patients in this study were based upon an average follow-up period of three years. Failures occurred in 3 per cent. In each case the knee was not stabilized. Fair results occurred in 17 per cent. One of these patients suffered mild pain; another had edema of the leg; and the remainder were improved, but they did not have as good a gait as they might have had because of improper equinus. Good results occurred in 80 per cent. These patients had a good gait, suffered no pain, and were pleased with the operative procedure.

In conclusion, it can be said that pantalar arthrodesis is a worth-while procedure, and, when carefully performed in properly selected patients, can be relied upon to give a uniformly satisfactory result.

REFERENCES

1. Steindler, A.: Treatment of Flail Ankle: Panastragaloid Arthrodesis, J. Bone & Joint Surg. **5**: 284, 1923.
2. Hamsa, W. R.: Panastragaloid Arthrodesis, J. Bone & Joint Surg. **18**: 732, 1936.

PATHOLOGY AND OPERATIVE CORRECTION OF FINGER DEFORMITIES DUE TO INJURIES AND CONTRACTURES OF THE EXTENSOR DIGITORUM TENDON

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THE operative correction of certain soft tissue deformities of the fingers presents difficulties and produces unsatisfactory results even when the most skilful technique is applied. This is true not only of cases in which the flexor surface structures are involved, but also, and perhaps, more frequently, when the injury is found on the extensor aspect of the digit.

The extensor digitorum communis tendon, as found over the dorsum of the fingers, was dissected by many anatomists. There was a controversy which lasted for many years about the minute structure and the function of the tendon. The gradual accumulation of knowledge on the physiology and anatomy of the hand is a very interesting subject but will not be dealt with in the present study.

It is my intention to present the anatomy of the extensor tendon in some detail, as this will permit better understanding of the four deformities which will be analyzed in this work; it will also help to grasp the principles of treatment for the correction of these deformities.

The description of the extensor tendon and its dependencies over the dorsum of the fingers is based on a personal investigation and, especially, on a recent study of these structures by R. Montant and A. Baumann of the University of Geneva, Switzerland. The two authors made serial cuts of fetal and adult fingers which were examined microscopically and then reconstructed on a larger scale. They also made clinical observations and dissections. This permitted the authors to establish more accurately the relationship between the extensor tendon, the interossei, and the lumbricals.

An anatomic investigation concerning muscle action can never be completed unless various methods of approach are used, such as electrical stimulation, clinical investigation on normal and abnormal muscles, and experiments on cadavera. The description of the physiology and anatomy of the extensor as given here is a result of such a study made by me and also based on combined efforts of numerous investigators who, beginning with Duchenne of Boulogne, laid the foundation of the exact physiology of the muscles of the body.

ANATOMY OF THE EXTENSOR APPARATUS OF THE FINGERS

The following is a description of the extensor tendon as given by French anatomists. The tendon of the extensor digitorum communis,

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arriving at the head of the corresponding metacarpal, maintains its course toward the base of the first phalanx; it bridges the metacarpophalangeal joint, forming the posterior capsule, and sends a strong insertion to the base of the first phalanx. The course of the tendon is continued over the insertion distally until it arrives at a point above the middle of the first phalanx where it divides into three distinct bands, one middle and two laterals (in relation to the axis of the finger). The middle band continues the course of the extensor tendon, bridges over the proximal interphalangeal joint, forming the posterior capsule for this joint, and inserts itself into the base of the middle phalanx. At this point the middle band terminates. The lateral bands run over the dorsolateral sides of the distal part of the first phalanx, then over the proximal part of the middle phalanx, and then gradually become more dorsal; the two bands unite at about the distal third of the middle phalanx. From here on this united band bridges the distal interphalangeal joint, forming again the posterior capsule, and finally inserts into the base of the distal phalanx. This terminal insertion sends up strands of fibers into the periosteum of the rest of the distal phalanx toward the matrix of the nail and to the skin. The extensor tendon, as mentioned above, forms the posterior capsule of the metacarpophalangeal, the proximal interphalangeal, and the distal interphalangeal joints. The terminal insertion of the extensor tendon enters into a complicated relation with the interossei and the lumbricals. This relation is very important, as only the combined action of all these muscles, as it is well known, contributes to a normal function of the fingers.

The interossei, arriving at the space between the metacarpal heads, form a flat tendon directed anteroposteriorly, which continues its course over the dorsolateral aspect of the capsule of the metacarpophalangeal joint; at the base of the proximal phalanx it divides into two parts: an anterior and posterior. The anterior forms a strong insertion to the lateral tubercule of the base of the proximal phalanx. The posterior continues distally, parallel to the lateral band of the extensor tendon and outside of it in relation to the long axis of the finger. Just above the metacarpophalangeal joint the interosseus tendon sends a flat membranous structure toward and over the middle band of the extensor tendon on the dorsum of the proximal phalanx. This membranous structure meets a similar structure formed by the interosseus on the other side of the finger and which covers the extensor tendon. It is called the expansion of the interossei and plays an importont role in flexion of the proximal phalanx. The rest of the interosseus tendon continues its course parallel to the lateral band of the extensor to the proximal interphalangeal joint; at this point it sends a few spiral fibers dorsal to the lateral band of the extensor tendon. The spiral bands cross obliquely the lateral bands of the extensor and become inserted to the median band at the base of the proximal phalanx. The interosseus

tendon continues its course further, running parallel to the lateral band and finally inserts itself, together with the lateral band, into the base of the distal phalanx as described above. The tendon of the lumbrical joins the interosseus and runs parallel to the interosseus and outside of it and the lateral band of the extensor tendon. The lumbricals are situated on the radial side of the tendons of the interossei.

Thus, the lateral bands which are observed on gross anatomical examination really consist of three distinct structures on the radial side of the fingers (the lateral bands of the extensor tendon, the interosseus and the lumbricals) and two on the ulnar side; namely, the lateral band of the extensor tendon and the interosseus (Fig. 1). It was found that the lumbrical unites with the interosseus distally to the interosseus expansion.

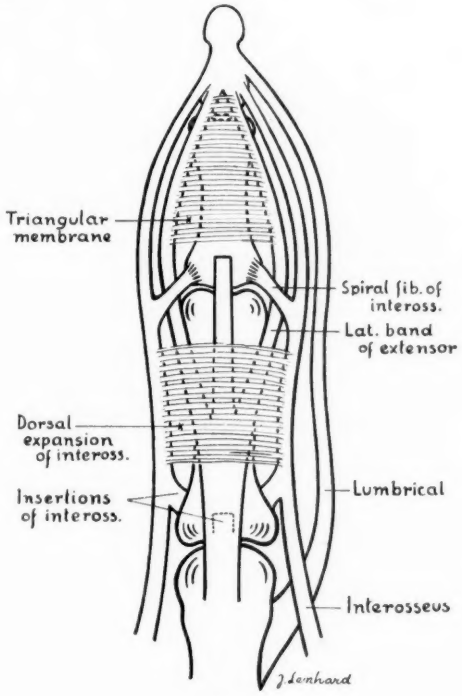

Fig. 1.

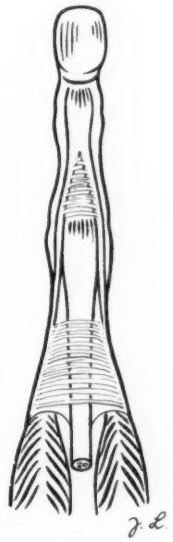

Fig. 2.

Fig. 1.—Schematic representation of the dorsal aspect of the extensor tendon with the tendons of the interosseus and lumbrical artificially separated. At the base of the first phalanx the insertion of the median band of the extensor tendon is shown in a broken line. In hyperextension of proximal phalanx the entire traction force of the extensor tendon is spent at this insertion.

Fig. 2.—A semischematic illustration of the triangular membrane on the dorsum of the middle phalanx.

Montant and Baumann called special attention to a structure which apparently is very important. They describe a very resistant, although very thin, membrane which they call the triangular membrane. This membrane is located at the dorsum of the middle phalanx between the lateral bands of the extensor (Figs. 1 and 2). This membrane prevents the lateral band from sliding toward the lateral sides of the finger when

flexion is produced at the proximal interphalangeal joint. The lateral bands are connected by fibers, perpendicular to the long axis of the finger, to the osteoperiosteal tunnel of the flexor tendons of the finger. These perpendicular fibers pull the lateral bands toward the volar surface. The balance between the intact triangular membrane and the perpendicular fibers maintains the normal position of the lateral bands over the dorsum of the middle phalanx. The description of the extensor

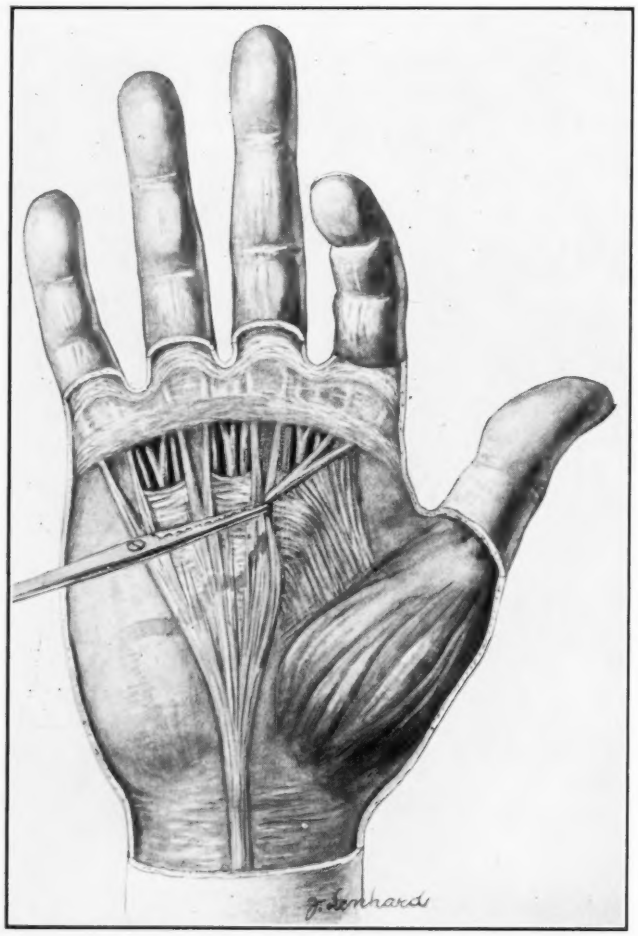

Fig. .3—Palmar fascia after removal of skin. Experimental traction on the longitudinal pretendinous band of the index shows flexion of the proximal and middle phalanges.

apparatus including the interossei and lumbricals, as given above, is considered to be the more generally encountered. It should be emphasized, however, that numerous variations affecting the volume of the interossei and lumbricals as well as the shape, insertion and nerve supply may be seen. It is well established that the extensor digitorum is supplied by the radial nerve, the interossei by the deep branch of the ulnar, and the lumbricals by the median for the two outer lumbricals and by the ulnar

for the inner lumbricals. It should be mentioned, however, that the interossei may also be supplied by branches of the radial as described by Rauber.

PHYSIOLOGY OF EXTENSOR APPARATUS

The present conception of the action of the extensor and the combined action of the interossei and lumbricals differs from the original description as given by Duchenne. Montant and Baumann summarize it in the following manner. When the finger is in full flexion, the extension of

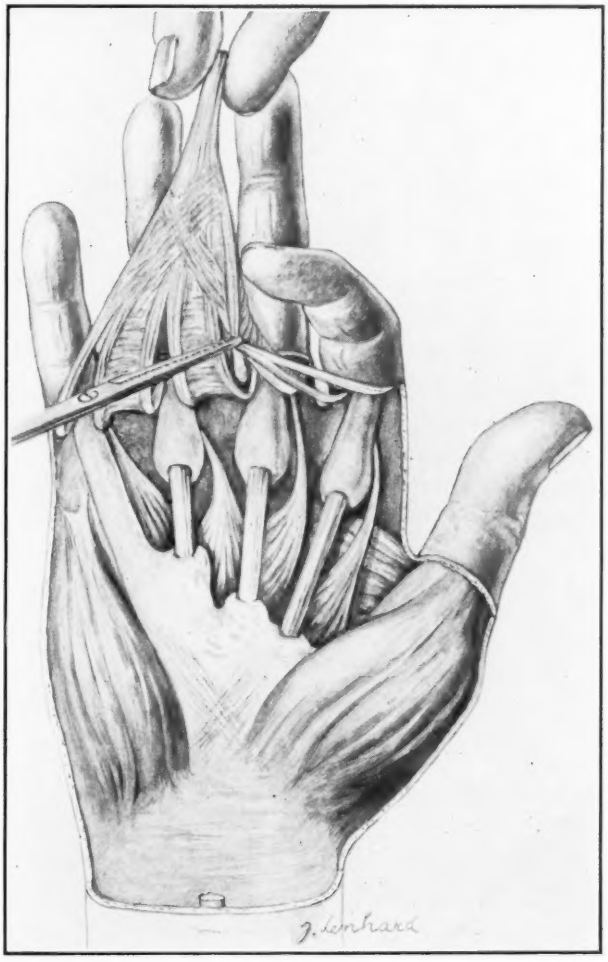

Fig. 4.—The palmar fascia separated from the underlying structures and lifted up. Experimental traction on the under surface of the longitudinal pretendinous band shows flexion of the proximal and middle phalanges.

the finger beginning in the distal phalanx is due to a simultaneous traction of the extensor and the interossei-lumbrical with a preponderance of the extensor tendon action. This preponderance continues up to the moment when the finger reaches the horizontal plane, when the action of the extensor is equal to the action of the interossei-lumbrical. When

the proximal phalanx hyperextends, the action of the extensor on the two distal phalanges is not exerted any more, as it is completely spent at the insertion of the extensor tendon to the base of the proximal phalanx. The extension of the two distal phalanges then is due entirely to the interossei-lumbrical combination.

When an analysis is made of injuries to the extensor and interossei, the variations in nerve supply must be kept in mind and also the newest conceptions of muscle action. It should not be forgotten that the extensor communis has also an abductor-adductor action on the fingers. Most of all, it must be remembered that on a cadaver, as well as on surgical exposure of the extensor communis tendon, traction of the tendon, when the finger is completely flexed, will produce a gradual extension, beginning with the distal interphalangeal joint, then the proximal interphalangeal, and then the metacarpophalangeal joints. When the metacarpophalangeal joint is hyperextended by the extensor communis, the two distal phalanges flex because the extensor communis spends all its force at the base of the proximal phalanx; the interossei-lumbrical then completes the extension of the two distal phalanges. When the metacarpophalangeal joint is flexed by the combined action of the interossei-lumbrical, the two distal phalanges can be easily extended by the extensor communis which does not spend its traction force on the base of the proximal phalanx.

In partial ulnar paralysis, where the proximal phalanx is kept hyperextended at the metacarpophalangeal joint and the two distal phalanges are kept flexed, the explanation of this deformity becomes simple if it is considered that the extensor tendon cannot exert any action on the two distal phalanges when it spends all its traction force on the base of the proximal phalanx and the interossei and lumbricals are inactive on account of the ulnar nerve paralysis.

CERTAIN PATHOLOGIC CONDITIONS OF THE EXTENSOR APPARATUS

Dupuytren's Contracture.—In a recent study made by me on the relation between the palmar aponeurosis and Dupuytren's contracture, it was found that anatomically the flexion of the fingers is due to the contracture of the longitudinal pretendinous bands of the palmar aponeurosis. The longitudinal pretendinous extensions of the palmar aponeurosis, as shown by many authors, extend into the fingers between the skin and the osteoperiosteal tunnels of the flexor tendons and insert to the lateral sides of the phalanges. Investigating the insertion of the longitudinal digital extensions, I found that they insert themselves not into the periosteum of the phalanges but are connected with the lateral bands of the extensor apparatus of the finger distal to the proximal interphalangeal joint. This is schematically shown in Fig. 6.

Figs. 3 and 4 show the palmar aponeurosis with its digital expansions and the effect produced by traction applied to a longitudinal pre-

tendinous band when the palmar aponeurosis is left intact (Fig. 3) and when the palmar aponeurosis is separated from the underlying tissues (Fig. 4) as reported in a previous communication. In Dupuytren's contracture the flexion contracture is usually pronounced at the proximal interphalangeal joint (Fig. 5). This marked flexion is apparently due to a contracture of the digital expansions of the palmar aponeurosis which is attached to the lateral bands of the extensor tendon. In considering the operative correction of Dupuytren's contracture, it is important, therefore, to remove the longitudinal pretendinous bands of the palmar aponeurosis from the palm of the hand and from the fingers and, especially, to separate these aponeurotic expansions from the lateral bands of the extensor tendon.

Fig. 5.—Dupuytren's contracture.

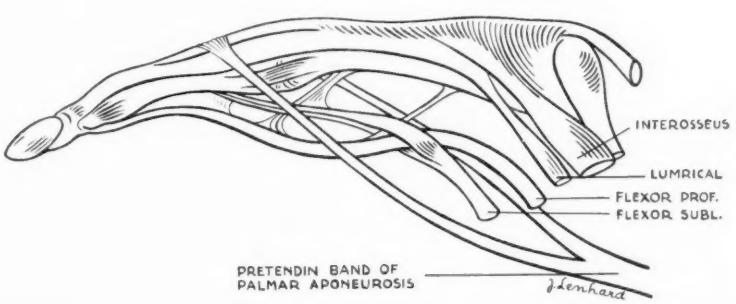

Fig. 6.—Digital expansions of the longitudinal pretendinous band of palmar aponeurosis connected with the lateral bands of the extensor digitorum tendon, as found by personal investigation and schematically represented. (Illustration partly redrawn from Toldt's *Atlas of Anatomy*, Berlin, 1921, Vrach Publishing Co.)

Mallet Finger or Baseball Finger.—A study of the so-called baseball finger convinced me that in the majority of cases the deformity is not due to an avulsion of the insertion of the extensor tendon from the base

of the distal phalanx. An examination of several of these cases immediately after the accident revealed that the area of acute pain was located over the middle of the dorsum of the middle phalanx. This area appeared slightly depressed as if the underlying structures had slipped away between the skin and the phalanx; a slight bulge was found at the distal end of the dorsum of the middle phalanx. These findings suggested the possibility of a different location of the tear. Indeed, on operation it was found that the tear of the extensor tendon was situated not at the base of the distal phalanx but over the middle or distal third of the dorsum of the middle phalanx, involving the two lateral bands and, at times, the triangular membrane, as schematically shown in Fig. 8. To repair this tear a procedure which is illustrated in Fig. 9 was used. The injured area is exposed through a U-incision with a distal base. The incision begins at the lateral side of the finger in line with the distal

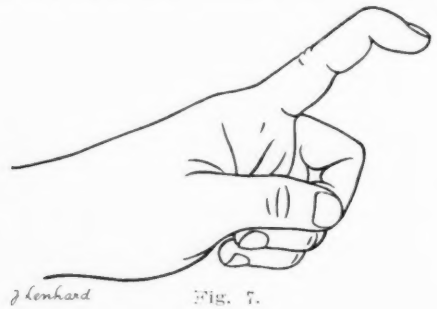

Fig. 7.

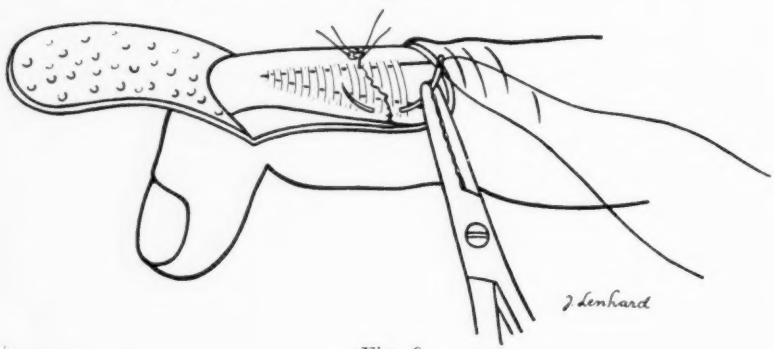

Fig. 9.

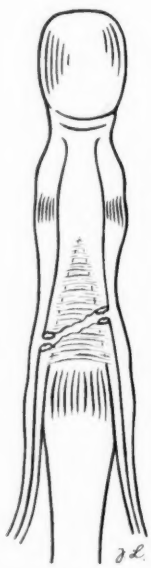

Fig. 8.

Fig. 7.—Baseball finger or mallet finger. Impossibility to extend the distal phalanx.

Fig. 8.—Pathology of baseball finger, consisting in a more-or-less oblique tear of the lateral bands of the extensor tendon and the triangular membrane between them.

Fig. 9.—Operative correction of mallet finger deformity. Description in text.

interphalangeal joint, runs parallel to the long axis of the finger at the junction of the lateral and dorsal lines of the finger. It rounds slightly distally to the proximal interphalangeal joint and comes out symmetrically on the other lateral side of the finger. The exposure usually shows the lateral bands and the above described triangular membrane, torn transversely over the dorsum of the middle phalanx. The repair consists in suturing the torn lateral bands as shown in the illustration.

The distal phalanx comes up in extension as soon as the torn lateral bands are sutured together. The finger is kept on a splint for three weeks, the hand being kept in neutral position and the metacarpophalangeal joint in slight flexion.

It is not denied that occasionally chip fractures of the base of the distal phalanx may occur, but these are due to a different type of injury and may heal on a hyperextension splint. It is advisable to x-ray the finger before the operative procedure is undertaken.

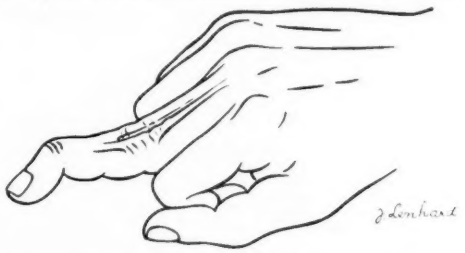

Fig. 10.—Flexion deformity of the distal phalanx combined with extension of the proximal interphalangeal joint.

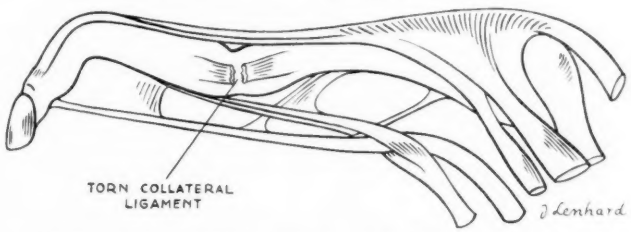

Fig. 11.—Pathology of the above-described deformity, consisting in a tear of the lateral ligaments of the proximal interphalangeal joint with a sliding toward the dorsum of the finger of the lateral bands of the extensor tendon.

Hyperextension of the Proximal Interphalangeal Joint.—

The fingers sometimes present a special deformity which follows an impact to the volar surface of the distal two phalanges. There is immediate pain over the volar surface of the proximal interphalangeal joint with swelling. Subsequently the patient develops an inability to flex the finger at the proximal interphalangeal joint after full extension of the fingers. A typical case was previously described as follows: The injured finger could be passively hyperextended to about 205° at the proximal interphalangeal joint. This finger could be actively hyperextended to about 190° at the same point. After full voluntary hyperextension of the finger, an attempt to flex all the fingers (to make a fist) resulted in normal flexion in all of the fingers except the middle one, which remained hyperextended at the proximal interphalangeal joint. The distal phalanx of this finger was the only one that flexed almost to 100°. Two distinct lateral cords of the collateral extensor tendon of the finger could be seen to be very tense under the skin of the dorsal aspect of the finger. No matter how hard the patient tried to flex this finger, it would remain hyperextended at the proximal interphalangeal joint. (Fig. 10.)

A personal investigation previously reported by me showed that this deformity was due to a tear of the lateral ligaments of the proximal interphalangeal joint and could be easily reproduced on a cadaver. The lateral bands of the extensor tendon, after an injury of this sort, are gradually retracted toward the dorsum of the finger. The sliding of these bands from their normal dorsolateral position into a dorsal position changes their function. When an extension of the finger is produced, the transverse axis of the proximal interphalangeal joint slips below the normal traction of the lateral extensor bands and a subsequent effort to flexion produces a hyperextension at the interphalangeal joint. Fig. 11 illustrates the pathology of this deformity.

The operative correction of this deformity can be obtained by a small lateral incision over the proximal interphalangeal joint on each side of the finger (Fig. 12). It consists in a repair of the torn collateral ligaments of the interphalangeal joint in cases where the collateral ligament can be easily recognized or in a reefing of the lateral volar part of the

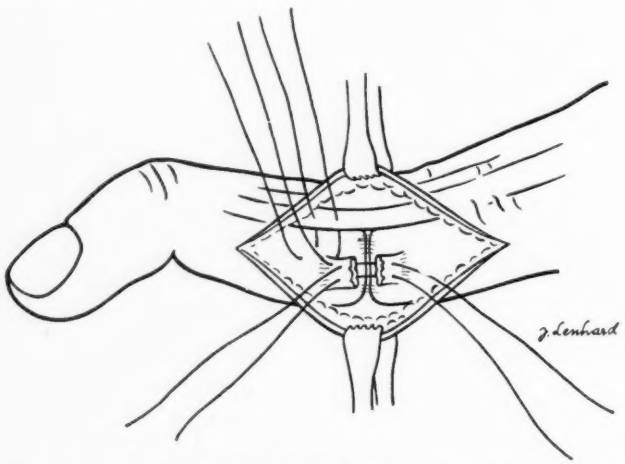

Fig. 12.—Operative correction of this deformity consisting in reparation of the lateral ligaments as proposed by the author previously, with an additional suture of the lateral bands to the lateral periosteum of the middle phalanx. This is done through a short incision over each side of the finger.

capsule of the interphalangeal joint in cases where the collateral ligament cannot be recognized. It is also necessary to bring the lateral bands of the extensor tendon from the dorsum of the finger to the lateral sides and suture them to the periosteums of the middle phalanx. After the closure of the wound, the finger is placed in slight flexion of all the joints and the hand in neutral position for three weeks.

Buttonhole Tear of the Extensor Apparatus.—This produces a flexion deformity of the proximal interphalangeal joint and an extension of the distal phalanx at the distal interphalangeal joint (Fig. 13). This deformity is best demonstrated when the patient, having all his fingers flexed, makes an effort to extend the fingers. The fingers will come up to normal extension, except the injured finger. The middle phalanx of

the injured finger remains flexed and the more the patient tries to extend it the more flexion is produced at the proximal interphalangeal joint. The distal phalanx hyperextends. This deformity is usually produced by a direct force applied to the dorsum of the middle phalanx or by a

Fig. 13.

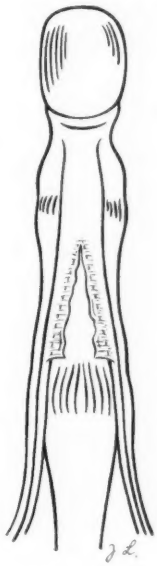

Fig. 14.

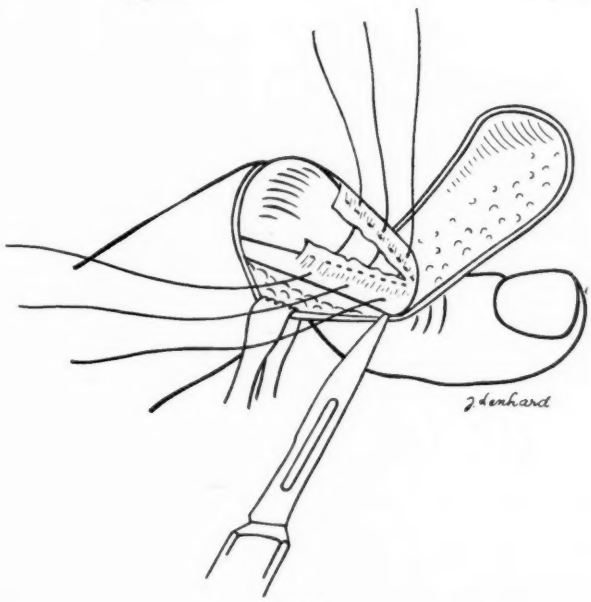

Fig. 15.

Fig. 13.—Buttonhole rupture of the extensor tendon with a typical flexion of the proximal interphalangeal joint and extension of the distal phalanx.

Fig. 14.—Pathology of the buttonhole rupture as shown by R. Montant and A. Baumann, consisting in a tear of the triangular membrane.

Fig. 15.—Operative correction of the buttonhole deformity as proposed by R. Montant and A. Baumann, consisting in a suture of the lateral bands over the dorsum of the middle phalanx with the author's addition of a longitudinal separation of the dislocated lateral bands from the lateral sides of the phalanx.

forced hyperflexion of the proximal interphalangeal joint while the finger is held in extension.

The mechanism of this deformity was described in detail by Montant and Baumann. They found that the deformity is due primarily to a longitudinal rupture of the triangular membrane which they described in detail and which unites the complex lateral bands of the extensor tendon over the dorsum of the middle phalanx. The lateral bands are held over the dorsum of the middle phalanx by this membrane which does not let them slip laterally under the action of the above mentioned connection of these bands with the osteoperiosteal tunnel of the flexor surface of the finger. The rupture of the triangular membrane permits a luxation of the lateral bands toward the volar surface of the fingers; the proximal interphalangeal joint becomes pinched between the lateral bands. The effort to extend a finger with such an injury produces traction on the dislocated lateral bands below the transverse axis of the proximal interphalangeal joint and thus produces flexion instead of the normal extension. (Fig. 14.)

The correction of this deformity can be obtained by suturing the lateral bands together over the dorsum of the middle phalanx, from the apex of the triangular membrane to a line just distal to the proximal interphalangeal joint as proposed by Montant and Baumann. Unfortunately the approximation of these two bands in old cases may prove to be a very difficult task. The lateral bands retract toward the volar surface and may adhere to the periosteum in their abnormal position. I found it, therefore, necessary to separate the lateral bands longitudinally from the periosteum as it is indicated in Fig. 15. A U-incision with a distal base as used for the deformity described before is advised; it gives the best exposure of the dorsum and the lateral sides of the phalanx and facilitates the operative procedure. After the operation the finger is kept in extension on a splint for three weeks, the hand being kept in neutral position and the metacarpophalangeal joint in slight flexion.

SUMMARY

The anatomy and physiology of the extensor apparatus of the fingers is presented. Four deformities of the fingers are described, showing their pathology and the operative correction.

REFERENCES

Auchincloss, H.: Surgery of the Hand, in Nelson's *Loose Leaf Surgery*, 1932, chap. V, vol. III.

Beevor, C. E.: The Croonian Lectures on Muscular Movements and their Representation in the Central Nervous System, Delivered before the R. C. of Physicians of London, June, 1903.

Deaver, J. B.: Surgical Anatomy of the Human Body, Philadelphia, 1926, P. Blakiston's Son & Co., plate CLXXXV, Vol. II, p. 228.

Duchenne, G. B. A.: Physiologie des Mouvements, Paris, 1867, J. B. Bailliere et Fils, pp. 162-206.

Kaplan, E. B.: Extension Deformities of the Proximal Interphalangeal Joints of the Fingers, J. Bone & Joint Surg. **18**: 781, 1936; with correction **19**: 1144, 1937.

Kaplan, E. B.: The Palmer Fascia in Connection With Dupuytren's Contracture, SURGERY **4**: 415, 1938.

Koch, S. L.: Disabilities of Hand Resulting From Loss of Joint Function, J. A. M. A. **104**: 30, 1935.

Mackenzie, C.: The Action of Muscles, London, 1930, H. K. Lewis.

Milch, H.: Button-Hole Rupture of the Extensor Tendon of the Finger, Am. J. Surg. **13**: 244, 1931.

Montant, R., and Baumann, A.: Anatomical Research on the System of the Extensor Tendon of the Fingers, Ann. d'anatomie path. **14**: 311, 1937.

Montant, R., and Baumann, A.: Rupture-luxation of the Extensor Apparatus of the Fingers at the First Interphalangeal Articulation, Rev. d'orthop. **25**: 5, 1938.

Poirier, P., and Charpy, A.: Traite d'Anatomie Humaine, Paris, 1912, Masson et Cie, vol. II.

Steindler, A.: Mechanics of Normal and Pathological Locomotion in Man, Baltimore, 1935, Charles C. Thomas, Publisher, p. 330.

DIETARY DEFICIENCIES IN SURGICAL PATIENTS

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DIETS commonly prescribed for dysfunction of the gastrointestinal tract are often unnecessarily rigid and in many instances actually are deficient in vitamins, minerals, and caloric value. Patients who have benefited by dietary measures are prone to rationalize that if dieting helps more dieting must certainly have added virtue. They may remain on stricter diets than physicians have intended and persist in these over periods ranging from weeks to years. If, as so often happens, these patients are plunged suddenly into a crisis by an acute exacerbation of their previous affliction or one related to it, they may exhibit alarming states of nutritional deficiencies. Vitamins are dissipated more rapidly when metabolism is increased or temperature elevated. A knowledge of vitamins and their relation to health and well-being is no longer confined to the research worker or faddist. The general public is becoming diet conscious and the restrictions imposed or elected by patients must be anticipated by both internists and surgeons if grave deficiency states are not overlooked to the great detriment of the patient. Operative risks may be lessened and postoperative convalescence greatly facilitated if optimum vitamin states are maintained. A brief review of the better known vitamins, their sources and uses, may be helpful at this point.

Fat-soluble vitamin A occurs in butter fat, egg yolk, animal fat, and many fish oils. It has been shown that carotene[1] of vegetables is the precursor of vitamin A, and it is thought that it is converted into vitamin A in the liver by the action of the enzymes. Deficiency of vitamin A produces a keratinization of epithelial surfaces. Columnar epithelium is changed to stratified epithelium. Cornification takes place with blocking of ducts of the glands present in the epithelial surface. Cyst formation and ulceration may follow. Vitamin A deficiency produces these changes in all epithelial surfaces. In the eye they give rise to xerosis, ulceration of lacrimal glands, and night blindness. Similar changes occur in the skin, respiratory tract, gastrointestinal tract, and the genitourinary tract. It has been suggested that, since epithelial surfaces are the first line of defense against infection, altered epithelial function in vitamin A deficiency lowers resistance to infection.[2]

Vitamin D is fat soluble and its sources are butter fat, animal fat, egg yolk, and fish oils. It controls calcium and phosphorous metabolism

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and its deficiency may cause the development of bone changes, especially rickets. The role of viosterol, calcium, and bile in the management of the hemorrhagic tendency in jaundice has been stressed by McNealy, Shapiro, and Melnick.[27] Osborne, Mendel, and Ferry[28] were first to call attention to the occurrence of urinary calculi in rats fed on diets deficient in fat-soluble vitamins, but the exact mechanism of stone formation under these conditions was not clear. More recently the work of Steiner, Zuger, and Kramer[29] has done much to clarify the part played by the changes in the lining epithelium of the urinary tract. Higgins[30] reports a marked decrease in the number of recurrences of renal calculi when a high vitamin A diet was instituted.

Most of our gastrointestinal diets contain adequate amounts of milk and cream so that a deficiency of vitamins A and D is not probable. The water-soluble vitamins B and C are more important in this discussion.

Vitamin B occurs most abundantly in yeast. Other sources are meat, glandular organs, and vegetables. There are apparently at least two factors in this vitamin: B_1 the antiberiberi factor; and B_2 (G), the antipellagra factor. Nerve lesions are common in deficiency states due to either of these factors. Deficiency of B_2 also produces mental changes, skin desquamation, and gastrointestinal disturbances. McCarrison[3] states that vitamin B deficiency causes gastrointestinal changes similar to colitis. Common symptoms are anorexia, weakness, vague pains, indigestion, and hypotonicity of the bowel. McCarrison states further that many of the gastrointestinal disorders of today are due to deficient and ill-balanced diets. He suggests that the health of the alimentary canal is dependent on vitamins B and C. Ravdin and Frazier[31] suggest that vitamin B complex be used to improve carbohydrate metabolism in the preoperative management of hyperthyroidism.

Vitamin C occurs in most fresh fruits and vegetables, our best sources being orange juice, tomato juice, and grapefruit juice. Deficiency of vitamin C causes scurvy. Clinically scurvy is characterized by hemorrhage from the skin (ecchymosis, typically perifollicular), mucous membranes, muscles, nerve sheaths, and periosteum. These hemorrhagic manifestations of vitamin C deficiency are due to an impairment of intercellular substance supporting the blood vessels. The endothelium of the capillaries is unable to form normal cement substance allowing diapedesis of red blood cells and loss of serum. This is particularly true of the blood channels supplying the pilosebaceous apparatus.[4] Bone changes also occur in scurvy due to a disturbance of the ossification rather than the calcification, as in rickets. Scorbutic patients also show a secondary anemia due to disturbance of hematopoiesis.

Methods are now available for the measurement of the state of vitamin C nutrition. In our determinations we have followed the method of Farmer and Abt.[5] Clinical levels of vitamin C nutrition have been

given by Ingalls and Warren.[6] Blood plasma levels of 1.0 to 2.0 mg. per cent are considered saturated. Normal is from 0.7 to 1.0 mg. per cent. Low normals run from 0.5 to 0.7 mg. per cent. Suboptimum levels range from 0.3 to 0.5 mg. per cent. Blood plasma levels of vitamin C between 0.15 and 0.3 mg. per cent are considered as asymptomatic scurvy and levels below 0.15 mg. per cent usually are found in cases of clinical scurvy.

Besides the hemorrhagic, bone, and blood changes commonly seen in scurvy, it has been shown that vitamin C also plays a role in resistance to infection. McConkey and Smith[7] found that, by feeding tuberculous sputum to animals, it could be shown that animals deficient in vitamin C rapidly developed extensive ulceration of the gastrointestinal tract and normal animals did not.

Recent work shows that there are two other less well-known vitamins that are definitely related to the bleeding tendency of patients. Vitamin K is a specific fat-soluble substance, the absence of which in the diet of chicks causes slow blood clotting (Dam[8]). Vitamin K occurs in hogs' liver, some fish, soy beans, and alfalfa (Dam and Schöenheyder[9]). Deficiency of vitamin K in human beings possibly causes some of the obscure hemorrhagic diseases. Neither lemon juice nor cevitamic acid alleviates the symptoms (McCay[10]). The other less well-known vitamin has been designated "citrin" or vitamin P. It occurs in lemons and red peppers. Deficiency of this factor causes increased permeability of capillaries.[11, 12]

DISCUSSION

Alvarez[13] reports the case of a woman with an ulcer who had bled until her hemoglobin was only 50 per cent. Because of fear, she stayed on a diet of milk, cream, and cereal for months. Her recovery undoubtedly was delayed because of vitamin and iron deficiency. Platt[26] reported 4 cases of clinical scurvy in adults. All were on restricted diets. Three of the cases were peptic ulcer patients. Ingalls and Warren[6] studied the blood plasma level of vitamin C in 20 patients with ulcers and showed that 90 per cent had low levels and 60 per cent had levels that were compatible with those of asymptomatic scurvy. Harris and Abbasy[14] studied the vitamin state of 74 medical and surgical cases. The average daily excretion of the entire group of 74 cases studied was only 8.9 mg. of cevitamic acid as compared with a normal urinary excretion of 13 mg. per day. In this study there were 12 cases of peptic ulcer and 7 other cases classed only as "dyspepsia." These 19 cases all fell below the average of the group with a daily urinary excretion of 5.6 mg. of cevitamic acid. In contrast, Elder and Emery[15] studied a series of ulcer and normal patients and found their diets to balance very closely.

It is evident that borderline states of nutritional instability are common. There is a wide zone between the state of the optimum nutrition and the level at which the classic symptoms of "diet deficiency states" develop.

Wolbach[16] has presented evidence that vitamin C is necessary for the proper growth and multiplication of all supporting tissue cells. It is also necessary for the formation of all intercellular substances which have collagen as their base and is as specifically related to soft tissue repair as is vitamin D to bone growth. Wolbach defines scurvy as an inability to produce and maintain intercellular substance. Proof of this is presented in his and Howe's study[17] of collagen in soft tissue repair. The necessity for optimal vitamin C nutrition in patients is obvious. With others, Smith and McConkey[18] showed that vitamin C deficiency in guinea pigs predisposes to the formation of gastric ulcers. On this finding they suggest the need for a vitamin C source in the diet of ulcer patients.

Following operative cases of peptic ulcer and carcinoma of the stomach, it is not uncommon to encounter cases which show delay in wound healing. Wolbach's work lends evidence in support of the importance of the role of vitamin C in this complication. Payne[19] has analyzed the cause of death in 51 patients who were subjected to operations on the stomach at St. Bartholomew's Hospital during the past sixteen years. Sixteen of these deaths were due to peritonitis; 12 of the 16 apparently originated at the site of the anastomosis where leakage occurred along the suture line. This was apparently due to almost complete lack of fibrin deposit. Lanman and Ingalls[20] reported the case of an infant who was operated upon for congenital atresia of the bowel. Postoperatively the child eviscerated and subsequently died. At post mortem the microscopic sections showed evidence of scurvy in the costochondral junctions and sections of the surgical wound showed numerous fibroblasts without the formation of collagen. This case prompted these authors to laparotomize a series of guinea pigs that were on a scorbutogenic diet and a control series maintained on a normal diet. Study of these two groups showed that on application of pressure the laparotomy wounds of the scorbutic group ruptured at a pressure level one-fourth as great as that required to rupture the wounds in the normal group. Study of the wounds of the scorbutic group revealed cyanotic, soft, friable wounds with an absence of collagen formation. These changes were similar to those reported in Wolbach's[16] study of tissue repair in the presence of scurvy.

In Ingalls and Warren's[6] study of asymptomatic scurvy and its relation to wound healing, they cite a patient with carcinoma of the pylorus who had been on a diet of milk, cream, and eggs for a period of six months. This patient's wound opened postoperatively and a secondary closure was done, but the patient died soon afterward. At the post-

mortem examination, the abdominal wound and the gastrojejunostomy presented a picture compatible with that found in the healing wound of a scorbutic patient.

With regard to peptic ulcer patients on medical management, there is evidence that a vitamin C deficiency may encourage continued bleeding. This hemorrhagic tendency may be due to an impairment of intercellular substance supporting mucosal cells and capillaries. Carlson and Rivers[21] presented a study of this phase of the problem. Lajos'[12] work with "citrin," commonly referred to as vitamin P, established its effect on resistance and permeability of capillaries and suggests that a deficiency state may be responsible for the continued bleeding of some peptic ulcers.

We recently undertook a routine study of our surgical cases which gave histories of prolonged dietary management. The findings in our series were so impressive that it prompts us to present this paper, which has as its principal purpose the outline of a scheme of management of patients who are candidates for, or have had, operations upon their gastrointestinal tracts.

In our study we have attempted to outline a dietary regimen that is balanced and affords adequate amounts of minerals, vitamins, and calories. Older diets seem to have shunned foods containing our main source of vitamin C because they were thought to contain too much free acid. In Kugelmass'[22] study he showed orange juice to have a pH of 3 and tomato juice to have a pH of 4; thus they contain very small amounts of free acid and can be considered fairly good buffers. Recently Dimmler and Power[23] studied the pH of orange juice and found it to vary from 3.63 to 3.99. Gastric juice following Ewald meals showed pH values varying from 1.12 to 7.07 with a mode of 1.6. Mixtures of one part of orange juice and four parts of gastric juice were studied both in vivo and in vitro. These mixtures showed pH values of 1.3 to 4.6. Thus, the orange juice lowered high gastric acidities and raised low gastric acidities. From this work it is reasonable to conclude that orange juice may be given safely to patients with peptic ulcer.

Preoperatively we have attempted to determine diets which fulfill the needs of the individual patients. It has been our plan to include those nonirritating, easily digested foods which are good sources of minerals, vitamins, complete proteins, and calories. Vitamin concentrates, such as cevitamic acid, brewers' yeast, and A.B.D. concentrates, are used because the quantity of food may have to be restricted. By their use, normal levels may be more quickly restored in the body. In the absence of free hydrochloric acid, as shown by gastric analysis, hydrochloric acid should be supplied, since it helps to decrease the amount of infection present in the stomach. Gastric cases showing obstruction should be adequately decompressed before surgery is attempted, to restore the stomach to as near normal as possible (McNealy and

Lichtenstein[24]). We have felt that material aspirated from the stomach during the period of decompression and restoration of positive pyloric balance might contain essential hormones, enzymes, or vital factors. To prevent their loss, we routinely administer the aspirated material to the patient by proctoclysis. It is usually added to the rectal drip of normal salt solution.

Postoperatively, we have kept patients in a state of fluid and chemical balance by adhering to the principles of water exchange as advocated by Coller, Dick, and Maddock.[25] During the first four days, postoperative patients are usually too ill to tolerate attempts to balance their diets. They ordinarily receive only 400 to 700 calories per day during this period and this is supplied almost entirely by glucose. By the fifth day, vitamin concentrates usually can be given by mouth. If, during the first four-day period, it seems imperative to augment vitamin C, this can be done parenterally. A study of the regimen we suggest shows that by the eighth postoperative day the caloric intake has reached 1,300 calories and by the end of two weeks the total caloric intake is nearly 2,000 calories and contains 56 gm. of protein, 1.1 gm. calcium, 1.3 gm. of phosphorus, and 13.7 mg. of iron. Even though this diet more than fulfills the requirements of most bed patients, it is steadily increased until on the fortieth day eight feedings with a maximum amount of five ounces are given. At this time the diet yields 2,825 calories and contains 86 gm. of protein, 2.5 gm. of calcium, 2.0 gm. phosphorus, 14.2 mg. of iron besides liberal amounts of each of the vitamins. If, however, chemical tests and physical findings indicate that the patient's depleted reserves have been fully restored, the diet is curtailed.

No restricted schedule of dietary management can be devised which will satisfy the requirements and the appetites of all the patients without modifications. For this reason, a considerable number of substitute foods have been included which may be used without materially changing the nutritional value of the diet. As milk, eggs, and cereals form the basis of this regimen, it is essential that the preparation and methods of serving be varied to prevent monotony.

SUGGESTED REGIMEN

General Directions.—

1. The patient is weighed on admission to the hospital and on the day before operation if his condition permits. He is weighed as soon after operation as his condition warrants and twice a week thereafter.

2. The postoperative administration of vitamin concentrates is begun on the fifth day. Cevitamic acid is given daily until the blood concentration reaches 1 mg. per 100 c.c. blood. Melvaron* is administered during the entire period of hospitalization.

*Supplied through courtesy of Eli Lilly Company. One fluid ounce contains: 28,000 U.S.P. units vitamin A, 8,000 U.S.P. units vitamin D, 1/12 International unit vitamin B, and 35 Sherman units B_2.

3. Iron therapy is begun on the tenth day and continued until the hemoglobin reaches 90 per cent.

4. The twenty-four-hour fluid intake is maintained at a minimum of 3,000 c.c. during the entire period of hospitalization.

5. Two egg whites are added to the fruit juice or milk mixture daily between the third and fifteenth postoperative days.

6. Cereal, in the form of pablum,* is added to the diet on the sixth day and continued until the twenty-third day, when farina, cornmeal, rice, and strained oatmeal may be substituted. Pablum is used because it is high in minerals and vitamins. It is prepared for serving by heating 5 gm. in 1 ounce milk mixture for each ounce of cereal ordered.

7. Puréed asparagus, carrots, peas, spinach, and squash are allowed on the thirteenth day. By the seventeenth day baked, baked-stuffed, boiled, creamed escalloped, and mashed potatoes may be substituted for the puréed vegetables.

8. Until the ninth day the eggs should be poached, soft boiled, or coddled. Thereafter, they may be served creamed or as soufflé or omelet.

9. Beginning on the ninth day, the following may be substituted for the milk mixture: eggnog, malted milk, cocoa, coco-malt, cal-e-malt, and banana milk. As soon as the patient regains his normal weight, the cream should be omitted in the preparation of these beverages.

10. The desserts allowed after the fourteenth day are ice cream, sherbets; banana, prune, or apricot whips; rice tapioca; bread, floating island, blanc mange, chocolate, butterscotch puddings; angelfood or sponge cake; plain or whipped gelatin.

11. Beginning on the fourteenth day, two ounces of cottage cheese may be substituted for one egg.

12. After the twenty-third day, baked noodles may be used in place of potatoes.

13. Beginning on the twenty-fifth day, canned peaches, pears, or peeled apricots may be given. Six days later peeled prunes, applesauce, baked apple (skin removed), ripe bananas, and grapefruit are added.

14. Tender boiled, broiled, or baked fish may be added on the thirty-first day.

REGIMEN

Preoperative Care.—

Diet:

As prescribed by attending surgeon. It should usually consist of a readily assimilable, nonresidue diet containing no seeds or coarse cellulose fibers.

Medication:

1. If patient can tolerate vitamin concentrates, administer ½ ounce melvaron, t.i.d.
2. Tablets of cevitamic acid; two 25 mg. tablets, t.i.d.

*Mead Johnson—analysis: protein, 15 per cent; fat, 3 per cent; carbohydrate, 70.8 per cent; moisture, 7 per cent; calcium, 0.78 per cent; phosphorus, 0.62 per cent; iron, 0.23 per cent; copper, 0.0013 per cent.

3. Jaundiced patients receive viosterol, 30 drops, t.i.d.; calcium, glucose, and bile salts if stools are acholic.

Laboratory Determinations:

Urinalysis.
Complete blood count.
Quantitative determination of blood cevitamic acid.
Jaundiced parents, Ivy bleeding time.

Weigh patient; record height.

Fluids:

Hydration and chemical equilibrium by salt solution, Hartman's or Ringer's solution.

Caloric complement by 5 per cent glucose solution intravenously or subcutaneously. The glucose to be given in distilled water unless the patient is chloride deficient.

Operative Day.—

Diet:

No food.
Fluids by mouth not given for the six hours preceding operation.

Medication:

Preoperative: Use of narcotics as ordered.
Postoperative: Morphine or its derivatives.

Fluids:

1,000 c.c. 5 per cent glucose solution administered subcutaneously in the loose axillary tissues during the operation.
1,000 c.c. 5 per cent glucose solution administered intravenously during the afternoon.
1,000 c.c. normal saline subcutaneously or intravenously during the night.
No enemas.

First Postoperative Day.—

Diet:

Nothing by mouth.

Medication:

Narcotics for pain or restlessness as ordered.

Fluids:

1,000 c.c. 5 per cent glucose solution subcutaneously during the morning.
1,000 c.c. normal salt solution intravenously during the afternoon.
1,000 c.c. 5 per cent glucose solution intravenously during the night.
Rectal tube for gas.

Second Postoperative Day.—

Diet:

Nothing by mouth except warm water and tea. Begin with teaspoonful administrations and, if tolerated, give at the rate of one ounce every hour.

Fluids:

5 per cent glucose solution and normal salt solution to supply at least 3,000 c.c. of fluid during the twenty-four-hour period.

Third Postoperative Day.—

Diet:

One ounce liquid every hour from 7:00 A.M. to 9:00 P.M. Water, tea, sweetened, cooked fruit juice (except grape), and milk mixture* (half milk, half cream) are allowed. Serve sugar with the tea.

*See page 54. Paragraph 5, General Directions.

Laboratory Tests:

Urinalysis.

Complete blood count and hemoglobin determination.

Quantitative cevitamic acid determination of blood.

Fluids:

5 per cent glucose solution and normal salt solution to supply 3,000 c.c. fluid during the twenty-four-hour period. The oral intake should be used in computing the minimum 3,000 c.c. requirement.

Fourth Postoperative Day.—

Diet:

Two ounces liquid every hour from 7:00 A.M. to 9:00 P.M.

Fluids:

Total minimum to be 3,000 c.c. in twenty-four hours.

Fifth Postoperative Day.—

Diet:

Two ounces liquid every hours from 7:00 A.M. to 9:00 P.M. Total fluids to be kept at 3,000 c.c.

Medication:

Begin the administration of vitamin concentrates.

½ ounce melvaron, t.i.d.

Two 25 mg. cevitamic acid tablets, t.i.d.

Sixth Postoperative Day.—

Cereal added.*

Seventh Postoperative Day.—

Same as above.

Eighth Postoperative Day.—

Diet:

Water may be given in 2-ounce quantities as the patient desires in addition to diet. Total fluids must be kept up to 3,000 c.c.

Ninth Postoperative Day.—

Diet:

Feedings every two hours from 7:00 A.M. to 9:00 P.M. Maximum quantity, 4 ounces per feeding.†

Fluids:

If oral water and liquid diet does not total 3,000 c.c. in twenty-four-hour period, salt solution by rectal or intravenous drip should be used.

Tenth Postoperative Day.—

Diet:

Feedings every two hours from 7:00 A.M. to 9:00 P.M. Maximum quantity, 4 ounces per feeding.

Medication:

Begin administration of iron.

Laboratory Tests:

Urinalysis.

Complete blood count and hemoglobin determination.

Quantitative cevitamic acid determination of blood.

Thirteenth Postoperative Day.—

Diet:

Feedings every two hours from 7:00 A.M. to 9:00 P.M. Maximum quantity, 5 ounces per feeding. Puréed vegetable allowed.‡

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*See page 54. Paragraph 6, General Directions.

†See page 54. Paragraphs 8 and 9, General Directions.

‡See page 54. Paragraph 7, General Directions.

Fourteenth Postoperative Day.—

Dessert allowed.*

Seventeenth Postoperative Day.—

Diet:

Feedings every two hours from 7:00 A.M. to 9:00 P.M. Maximum quantity, 6 ounces per feeding.

Laboratory Tests:

Urinalysis.
Complete blood count and hemoglobin determination.
Quantitative cevitamic acid determination of blood.

Twenty-First Postoperative Day.—

Diet:

Feedings every two hours from 7:00 A.M. to 9:00 P.M. Maximum quantity, 8 ounces per feeding.

Twenty-third Postoperative Day.—

Diet:

Feedings every three hours from 7:00 A.M. to 9:00 P.M. Maximum quantity, 9 ounces per feeding.†

Twenty-fourth Postoperative Day.—

Diet:

Feedings every three hours from 7:00 A.M. to 9:00 P.M. Maximum quantity, 9 ounces per feeding.

Laboratory Tests:

Urinalysis.
Complete blood count and hemoglobin determination.
Quantitative cevitamic acid determination of blood.

Twenty-Fifth Postoperative Day.—

Diet:

Feedings every three hours from 7:00 A.M. to 9:00 P.M. Maximum quantity, 10 ounces per feeding.‡

Twenty-Ninth Postoperative Day.—

Diet:

Feedings every three hours from 7:00 A.M. to 9:00 P.M. Maximum quantity, 12 ounces per feeding.

Thirty-First Postoperative Day.—

Diet:

Feedings every three hours from 7:00 A.M. to 9:00 P.M. Maximum quantity, 14 ounces per feeding.§

Laboratory Tests:

Urinalysis.
Complete blood count and hemoglobin determination.
Quantitative cevitamic acid determination of blood.

Thirty-Sixth Postoperative Day.—

Diet:

Feedings every three hours from 7:00 A.M. to 9:00 P.M. Maximum quantity, 16 ounces per feeding.

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*See page 54. Paragraphs 10 and 11, General Directions.
†See page 54. Paragraph 12, General Directions.
‡See page 54. Paragraph 13, General Directions.
§See page 54. Paragraph 14, General Directions.

TABLE I

HOURLY DIET REGIMEN

POSTOPERATIVE DAY MAXIMUM QUANTITY	3RD DAY 1 OZ. EVERY HOUR	4TH AND 5TH DAYS 2 OZ. EVERY HOUR	6TH AND 7TH DAYS 2 OZ. EVERY HOUR	8TH DAY 2 OZ. EVERY HOUR
7 A.M.	1 oz. fruit juice	2 oz. fruit juice	2 oz. fruit juice	2 oz. fruit juice
8 A.M.	1 oz. water or tea	2 oz. water or tea	2 oz. water or tea	2 oz. milk mixture
9 A.M.	1 oz. milk mixture	2 oz. milk mixture	1 oz. milk mixture 1 oz. cereal	1 oz. milk mixture 1 oz. cereal
10 A.M.	1 oz. fruit juice	2 oz. fruit juice	2 oz. fruit juice	2 oz. fruit juice
11 A.M.	1 oz. water or tea	2 oz. water or tea	2 oz. water or tea	2 oz. milk mixture
12 M.	1 oz. milk mixture	1 oz. milk mixture 1 egg	1 oz. milk mixture 1 egg	1 oz. milk mixture 1 egg
1 P.M.	1 oz. water or tea	2 oz. water or tea	2 oz. water or tea	2 oz. milk mixture
2 P.M.	1 oz. fruit juice	2 oz. fruit juice	2 oz. fruit juice	2 oz. fruit juice
3 P.M.	1 oz. water or tea	2 oz. water or tea	2 oz. water or tea	2 oz. milk mixture
4 P.M.	1 oz. milk mixture	2 oz. milk mixture	1 oz. milk mixture 1 egg	1 oz. milk mixture 1 egg
5 P.M.	1 oz. water or tea	2 oz. water or tea	2 oz. water or tea	2 oz. milk mixture
6 P.M.	1 oz. fruit juice	2 oz. fruit juice	2 oz. fruit juice	2 oz. milk mixture
7 P.M.	1 oz. water or tea	2 oz. water or tea	2 oz. water or tea	2 oz. milk mixture
8 P.M.	1 oz. milk mixture	2 oz. milk mixture	2 oz. milk mixture	2 oz. fruit juice
9 P.M.	1 oz. water or tea	2 oz. water or tea	2 oz. water or tea	2 oz. milk mixture

Thirty-Eighth Postoperative Day.—

Diet:

Feedings every three hours from 7:00 A.M. to 9:00 P.M. Maximum quantity, 16 ounces per feeding.

Laboratory Tests:

Complete blood count and hemoglobin determination.
Quantitative cevitamic acid determination of blood.

CASE EXAMPLES

CASE 1.—Patient F. T., male, aged 55 years. This patient gave an ulcer history of eight years' duration. He had been placed on Sippy management but only stayed on management as long as symptoms were present. He was employed as a cook and found it difficult to eat meals regularly. He developed the habit of gulping his food while working. His usual daily intake consisted of ice-cold milk, French fried potatoes, meat, and a little fruit.

TABLE I.—CONT'D

POSTOPERATIVE DAY	9TH AND 10TH DAYS	11TH AND 12TH DAYS	13TH AND 14TH DAYS
MAXIMUM QUANTITY	4 OZ. EVERY 2 HR.	4 OZ. EVERY 2 HR.	5 OZ. EVERY 2 HR.
7 A.M.	2 oz. cereal 1 oz. milk mixture 1 egg	2 oz. cereal 1 oz. cream 1 egg	1 egg 1 oz. cream 1 strip bacon 2 oz. cereal
9 A.M.	4 oz. orange juice	4 oz. orange juice	5 oz. orange juice
11 A.M.	4 oz. milk mixture	4 oz. milk mixture	2 oz. cream 3 oz. cereal
1 P.M.	1 soft cooked egg 2 oz. cereal 1 oz. milk mixture	1 soft cooked egg 2 oz. cereal 1 oz. cream	1 soft cooked egg 2 oz. puréed vegetable 2 oz. milk mixture 10 gm. butter—in vegetable
3 P.M.	4 oz. milk mixture	4 oz. ice cream, custard, or jello	4 oz. ice cream, custard, or jello
5 P.M.	1 soft cooked egg 3 oz. milk mixture	1 soft cooked egg 3 oz. milk mixture	1 soft cooked egg 4 oz. milk mixture
7 P.M.	4 oz. fruit juice	4 oz. fruit juice	5 oz. fruit juice
9 P.M.	4 oz. milk mixture	4 oz. milk mixture	5 oz. milk mixture

In January he developed a recurrence of symptoms and bleeding from the ulcer occurred. His symptoms and bleeding were refractory to a modified Sippy regimen and to mucin treatment. His weight was 104 pounds; hemoglobin, 60 per cent; R.B.C., 2.99; and blood cevitamic acid was 0.44 mg. per cent.

At operation a Polya type partial resection was performed. Convalescence was good except for a mild respiratory infection. The patient experienced no difficulty in taking the regular postoperative feedings provided the eggs were incorporated in the beverages. On discharge from the hospital weight was 105 pounds; hemoglobin, 64 per cent; R.B.C., 3.6; and blood cevitamic acid was 0.84 mg. per cent. Surgical wound was clean and well healed.

CASE 2.—Patient F. K., female, aged 47 years. This patient gave a history of fulness, belching, and distention for eight weeks, but she had been unable to retain food for four weeks prior to admission because of severe nausea and vomiting. There had been a weight loss of twenty pounds. Her weight was 105 pounds; hemoglobin, 93 per cent; R.B.C., 4.6; blood cevitamic acid was 0.16 mg. per cent.

Workup showed a pyloric obstruction due to carcinoma of the stomach. She was placed on our regimen and prepared for surgery. At operation a large infiltrating carcinoma was found. A Polya type subtotal gastric resection was performed.

Postoperatively the regimen was well tolerated. Patient vomited only once, that being on the fifth postoperative day. Convalescence was very smooth. Weight increased to 111½ pounds; hemoglobin was 80 per cent; R.B.C., 4.5; blood cevitamic acid was 1.56 mg. per cent. Operative wound was clean and completely healed.

CASE 3.—Patient F. H., female, aged 46 years. This patient was acutely ill when first seen. She gave a history of chronic gall-bladder disease over a period of sixteen years; present attack was of ten days' duration; severe right upper quadrant pain had been accompanied by such persistent vomiting that she had been unable to retain food. Temperature ranged from 100 to 102° F. A large extremely tender mass was present in the right upper quadrant and it extended almost to the crest of the ilium. Icterus index was 40 and blood plasma cevitamic acid was 0.38 mg. per cent. Hemoglobin was 90 per cent and R.B.C., 5; W.B.C. was 18,700.

TABLE I.—CONT'D

POSTOPERATIVE DAY	15TH AND 16TH DAYS	17TH AND 18TH DAYS	19TH AND 20TH DAYS	21ST AND 22ND DAYS
MAXIMUM FEEDING	5 OZ.	6 OZ.	6 OZ.	7 OZ.
NO. OF FEEDINGS	8	8	8	8
7 A.M.	1 slice toast 1 square butter 1 oz. cream 1 oz. cereal 1 egg	1 slice toast 1 square butter 1 oz. cream 2 oz. cereal 1 egg	1 slice toast 1 square butter 1 oz. cream 2 oz. cereal 1 egg	1 slice toast 1 square butter 2 oz. cream 2 oz. cereal 1 egg
9 A.M.	5 oz. orange juice	6 oz. orange juice	6 oz. orange juice	6 oz. orange juice
11 A.M.	2 oz. cream 3 oz. cereal	6 oz. cream soup 3 crackers	6 oz. cream soup 3 crackers	6 oz. cream soup 3 crackers
1 P.M.	1 strip bacon 1 egg 2 oz. puréed vegetable 1 square butter	1 strip bacon 1 egg 2 oz. puréed vegetable or potato 1 square butter 1 oz. cream	1 slice toast 2 oz. dessert 2 oz. pureéd vegetable or potato 2 squares butter 1 oz. cream	1 slice toast 3 oz. dessert 2 oz. puréed vegetable or potato 2 squares butter 1 oz. cream
3 P.M.	4 oz. dessert	4 oz. dessert	4 oz. dessert	4 oz. dessert
5 P.M.	1 egg 2 oz. milk mixture 1 slice toast 1 square butter	1 egg 3 oz. milk mixture 1 slice toast 1 square butter	1 egg 3 oz. milk mixture 1 slice toast 1 square butter	1 egg 4 oz. milk mixture 1 slice toast 1 square butter
7 P.M.	5 oz. beverage	6 oz. beverage	6 oz. beverage	7 oz. beverage
9 P.M.	5 oz. beverage	6 oz. beverage	6 oz. beverage	7 oz. beverage

On conservative treatment the gall bladder mass disappeared and the patient's condition improved markedly, except that R.B.C. fell to 3.6 and Hb. 54 per cent. At time of operation R.B.C. was 4 and Hb. 64 per cent. Blood cevitamic acid was 0.88 mg. per cent. A much thickened gall bladder containing stones was removed and a cholecystocolic fistula was closed. Convalescence was excellent and laparotomy wound healed without signs of infection. Regimen was well tolerated.

CASE 4.—Patient W. M., male, aged 40 years. This was a case of chronic peptic ulcer of four years' duration. Patient had been treated for peptic ulcers in 1934 but had followed his diet only a short time before he resumed his former eating habits.

For ten days prior to admittance to the hospital he had symptoms of severe pain and repeated vomiting. Weight was 101 pounds; R.B.C., 4.7; hemoglobin, 87 per cent; and W.B.C., 11,800. Gastric analysis showed free acid of 64 and a total of 94. Blood cevitamic acid was 0.32 mg. per cent.

This case appeared to be one of a penetrating ulcer with some pyloric obstruction. At operation a walled-off perforated ulcer was found and a Polya type resection was performed. Postoperatively patient ran a stormy course; he had considerable fever, developed a diarrhea, and had an infected wound. Because of these complications, it was difficult to meet optimum nutritional requirements. On discharge, patient weighed 83 pounds; hemoglobin was 64 per cent; R.B.C., 4; blood cevitamic acid, 0.4 mg. per cent. We have felt that, though the result in this patient was far from ideal, it might have been worse if no attempt had been made to supply deficient food factors.

TABLE I.—CONT'D

POSTOPERATIVE DAY	23RD AND 24TH DAYS	25TH, 26TH, 27TH, AND 28TH DAYS	29TH AND 30TH DAYS
MAXIMUM FEEDING	9 OZ.	10 OZ.	12 OZ.
NO. OF FEEDINGS	6	6	6
7 A.M.	1 slice toast 1 square butter 2 oz. cream 1 oz. milk mixture 3 oz. cereal 1 egg	1 slice toast 1 square butter 2 ounces cream 1 oz. milk mixture 4 oz. cereal 1 egg	1 slice toast 1 square butter 2 oz. cream 1 oz. milk mixture 4 oz. cereal 1 egg or 3 strips bacon
10 A.M.	6 oz. orange juice	6 oz. orange juice	6 oz. orange juice 1 slice bread 1 square butter
12 M.	6 oz. cream soup 3 crackers 2 oz. baked noodle or potatoes 1 square butter 2 strips bacon	6 oz. cream soup 3 crackers 2 oz. baked noodles or potatoes 1 square butter 2 oz. canned peaches, pears, or peeled apricots	6 oz. cream soup 3 crackers 2 oz. boiled or broiled chicken or fish 2 squares butter 2 oz. canned pears, peaches, or peeled apricots
3 P.M.	4 oz. dessert 5 oz. beverage	4 oz. dessert 6 oz. beverage	4 oz. dessert 6 oz. beverage
6 P.M.	1 egg 2 oz. puréed vegetable 1 square butter 4 oz. milk mixture or fruit juice	1 egg 2 oz. puréed vegetable 1 square butter 3 oz. dessert 2 oz. milk mixture	2 oz. puréed vegetable or potato 1 square butter 3 oz. dessert 4 oz. milk mixture
9 P.M.	9 oz. beverage	9 oz. beverage	9 oz. beverage

CASE 5.—L. F., female, aged 31 years. This patient gave a history of having a pelvic infection in 1929; in 1937 she was operated upon twice because she presented symptoms of a partial intestinal obstruction. When seen, she presented evidence of a severe nutritional disturbance as a result of the old pelvic infection and bowel interference from adhesions. Weight was 75 pounds; Hb., 44 per cent; and R.B.C., 2.6. Blood cevitamic acid was 0.44 mg. per cent.

This patient was put on a high caloric antianemic diet supplemented by vitamin concentrates and in a period of twenty-six days weight had increased to 85 pounds; Hb. was 55 per cent and R.B.C., 2.8. Cevitamic acid level was 1.08. Patient was much improved and was not operated upon.

CASE 6.—H. S., female, aged 70 years. This was a case of carcinoma of the stomach in an elderly female. Weight was 119 pounds; Hb., 40 per cent; R.B.C., 2.8; and blood cevitamic acid, 1.04 mg. per cent. Stomach was decompressed and patient given three transfusions prior to operation. At this time Hb. was 76 per cent and R.B.C., 5. A Polya resection was performed; postoperative course was fair except for a low-grade wound infection. Regimen was well tolerated. At time of discharge on thirty-sixth postoperative day, weight was 114 pounds; Hb., 65 per cent; R.B.C., 4.3; and blood cevitamic acid, 0.96 mg. per cent.

CASE 7.—Patient R. F., female, aged 36 years. This patient was acutely ill when first seen. She was markedly dehydrated; gums were spongy and bled freely; body was covered with perifollicular ecchymoses. The extremities were covered with perifollicular hemorrhages and the legs also showed periosteal hemor-

TABLE I.—CONT'D

POSTOPERATIVE DAY	31ST–35TH DAYS	36TH–40TH DAYS
MAXIMUM FEEDING	14 OZ.	16 OZ.
NO. OF FEEDINGS	6	6
7 A.M.	1 oz. fruit 2 slices toast 2 squares butter 2 oz. cream 4 oz. cereal 1 egg or 3 strips bacon 2 oz. milk	3 oz. fruit 2 slices toast 2 squares butter 2 oz. cream 4 oz. cereal 1 egg or 3 strips bacon 2 oz. milk
10 A.M.	6 oz. orange juice 1 slice bread 1 square butter	6 oz. orange juice 1 slice bread 1 square butter
12 M.	6 oz. cream soup 3 crackers 1 egg or substitute 2 oz. puréed vegetable 2 squares butter 1 slice bread 2 oz. fruit	6 oz. cream soup 3 crackers 1 egg or substitute 2 oz. puréed vegetable 2 squares butter 2 slices bread 3 oz. fruit
3 P.M.	4 oz. dessert 8 oz. beverage	4 oz. dessert 8 oz. beverage
6 P.M.	2 oz. puréed vegetable 1 slice bread 2 squares butter 1 egg or substitute 3 oz. fruit or dessert 6 oz. beverage	2 oz. puréed vegetable 2 slices bread 2 squares butter 1 egg or substitute 3 oz. fruit or dessert 6 oz. beverage
9 P.M.	8 oz. beverage	8 oz. beverage

rhages. The abdomen was markedly distended and a distinct bowel pattern was visible through the abdominal wall. Rectally, a hard, constricting, infiltrating mass was felt.

A history of bowel disturbance and loss of 30 pounds in weight over a period of six months was elicited. The patient was a religious woman who had not sought the advise of a physician but had "dieted" herself. Her diet consisted of milk and tea.

Blood cevitamic acid levels on two determinations showed values of 0.06 mg. per cent and 0.12 mg. per cent. Attempt at conservative decompression was unsuccessful and a cecostomy was performed after water and chemical balance was restored to as near normal as was possible. This patient expired three days later.

This case of carcinoma of the rectum, intestinal obstruction, and scurvy illustrates dramatically the high degree of deficiency disease that is possible in neglected patients.

CONCLUSIONS

From the evidence presented, it is apparent that borderline nutritional disturbances are common in many gastrointestinal conditions, especially in cases of peptic ulcer and carcinoma of the stomach. Deficiency of vitamins A, B, and C may initiate or perpetuate ulcerative lesions of the gastrointestinal tract. The health of the alimentary canal is especially dependent on adequate amounts of the vitamins B and C. Vita-

TABLE II

FOOD VALUES, POSTOPERATIVE DIET

	C	P	F	CALORIES	CA	P	FE
Operative day	100			400			
First postoperative day	100			400			
Second postoperative day	100			400			
Third postoperative day	141	3.9	14.4	718	.128	.10	.24
Fourth postoperative day	81	22.0	30.6	702	.267	.27	1.74
Fifth postoperative day	81	22.0	30.6	702	.267	.27	1.74
Sixth postoperative day	83	28.5	32.3	752	.307	.271	4.44
Seventh postoperative day	83	28.5	32.3	752	.307	.271	4.44
Eighth postoperative day	117	38.7	71.5	1,336	.691	.671	5.16
Ninth postoperative day	89	51.5	87.8	1,407	.969	1.121	11.38
Tenth postoperative day	89	51.5	87.8	1,407	.969	1.121	11.38
Eleventh postoperative day	103	51.7	92.7	1,563	.931	1.113	11.39
Twelfth postoperative day	103	51.7	92.7	1,563	.931	1.113	11.39
Thirteenth postoperative day	124	56.9	131.5	1,952	1.136	1.329	13.73
Fourteenth postoperative day	124	56.9	131.5	1,952	1.136	1.329	13.73
15th and 16th postoperative days	100.0	49.6	141.0	1,880	1.035	1.278	12.09
17th and 18th postoperative days	108.9	54.0	144.7	1,963	1.870	1.401	9.89
19th and 20th postoperative days	126.4	49.6	145.7	2,031	1.879	1.357	8.71
21st and 22nd postoperative days	132.4	53.8	165.3	2,242	2.023	1.473	9.04
23rd and 24th postoperative days	122.1	48.9	144.9	1,986	1.876	1.352	10.26
25th, 26th, 27th, 28th postoperative days	156.6	56.1	142.9	2,173	2.036	1.494	11.01
29th and 30th postoperative days	189.0	69.3	175.2	2,613	2.177	1.674	11.07
31st-35th postoperative days	217.0	82.6	160.2	2,650	2.561	2.012	14.28
36th-40th postoperative days	256.4	86.3	160.4	2,825	2.571	2.048	14.28

min C has a definite effect on fibroplastic healing and general reparative processes and adequate amounts are necessary to insure normal postoperative healing. Vitamin D in the form of viosterol has been shown to influence favorably the hemorrhagic tendency in jaundice when given in with bile. It is also suggested that deficiency of vitamin C and the two lesser known vitamins, K and P, may be important in cases that show continued bleeding. The administration of orange juice has no deleterious effect on cases of high acidity, but rather lowers the acidity. Vitamin C also may be supplied in the form of cevitamic acid tablets and in cases that have marked pyloric obstruction it may be administered parenterally.

In the diets we are presenting, we have attempted to balance the mineral and vitamin content and postoperatively we are supplying the patient with a more adequate caloric intake. In our cases the regimen has been well tolerated and we feel that the well-being of patients has been improved under this management.

REFERENCES

1. Moore, T.: Vitamin A and Carotene, Biochem. J. **24**: 692-702, 1930.
2. Mellanby, M., and Pottison, C. L.: Remarks on Influence of Cereal-Free Diet Rich in Vitamin D and Calcium, Brit. M. J. **1**: 507-510, 1932.
3. McCarrison, R.: Studies in Deficiency Disease, London, 1921. Henry Frowder and Hodder & Stroughton, Oxford Medical Publications.
4. Keil, H.: Follicular Lesions of Vitamin A and C Deficiency, Am. J. Digest. Dis. & Nutrition **5**: 40-48, 1938.
5. Farmer, C. J., and Abt, A. F.: Ascorbic Acid Content of Blood, Proc. Soc. Exper. Biol. & Med. **32**: 1625-1629, 1935.
6. Ingalls, T. H., and Warren, H. A.: Asymptomatic Scurvy. Its Relation to Wound Healing and Its Incidence in Patients With Peptic Ulcer, New England J. Med. **217**: 443-446, 1937.
7. McConkey, M., and Smith, D. T.: Relation of Vitamin C Deficiency to Intestinal Tuberculosis in Guinea Pigs, J. Exper. Med. **58**: 503-512, 1933.
8. Dam, H.: Hemorrhages in Chicks Reared on Artificial Diets: New Deficiency Disease, Nature, London **133**: 909-910, 1934.
9. Dam, H., and Schöenheyder, F.: Deficiency Disease in Chicks Resembling Scurvy, Biochem. J. **28**: 1355-1359, 1934.
10. McCay, C. M.: Less Well-Known Vitamins, J. A. M. A. **110**: 1441-1446, 1938.
11. Ruszniak, S., and Szent-Gyorgyi, A.: Vitamin P, Nature, London **138**: 27, 1936.
12. Lajos, S.: Clinical Experiences With "Citrin" (Vitamin P), Klin. Wchnschr. **16**: 1615-1617, 1937.
13. Alvarez, W. C.: Some Dangers Involved in Leaving Ulcer Patients Too Long on a Strict Diet, Am. J. Digest. Dis. & Nutrition **4**: 455-456, 1937.
14. Harris, L. J., and Abbasy, M. A.: Vitamin C Reserves of Subject of the Voluntary Hospital Class, Lancet **1**: 1488-1490, 1936.
15. Elder, M., and Emery, E. S.: Food Habits of Patients With Peptic Ulcer, Am. J. Digest. Dis. & Nutrition **4**: 493-494, 1937.
16. Wolbach, S. B.: Vitamin C and the Formation of Intercellular Material, New England J. Med. **215**: 1158-1159, 1936.
17. Wolbach, S. B., and Howe, P. R.: Intercellular Substances in Experimental Scorbutus, Arch. Path. **1**: 1-24, 1926.
18. Smith, D. T., and McConkey, M.: Peptic Ulcers: Occurrence in Guinea Pigs Fed on Diet Deficient in Vitamin C, Arch. Int. Med. **51**: 413-426, 1933.
19. Payne, R.: Post-Mortem Findings After Partial Gastrectomy, St. Barth. Hosp. Rep. **69**: 191-198.
20. Lanman, T. A., and Ingalls, T. A.: Vitamin C Deficiency and Wound Healing, Ann. Surg. **105**: 616-625, 1937.
21. Carlson, L. A., and Rivers, A. B.: Clinical Consideration of Defensive Factors of Tissues in Etiology of Peptic Ulcer, Rev. Gastroenterol. **4**: 96-101, 1937.
22. Kugelmass, I. N.: Acid-Base Value and Assimilability of Fruit Juices, Am. J. Digest. Dis. & Nutrition **2**: 242-243, 1935.
23. Dimmler, Charles, Jr., and Power, M. H.: Effect of Orange Juice on Gastric Acidity, Am. J. Digest. Dis. & Nutrition **5**: 86-87, 1938.
24. McNealy, R. W., and Lichtenstein, M. E.: Gastro-Jejunostomy—Pre-Operative Decompression, Surg., Gynec. & Obst. **63**: 96-98, 1936.
25. Coller, F. A., Dick, V. S., and Maddock, W. G.: Maintenance of Normal Water Exchange With Intravenous Fluids, J. A. M. A. **107**: 1522-1527, 1936.
26. Platt, R.: Scurvy as Result of Dietetic Treatment, Lancet **231**: 366-367, 1936.
27. McNealy, R. W., Shapiro, P. F., and Melnick, P.: The Effect of Viosterol in Jaundice, Surg., Gynec. & Obst. **60**: 785-801, 1935.
28. For references to the work in this field, see report of the Council on Pharmacy and Chemistry, Vitamin A, and Urinary Lithiasis, J. A. M. A. **105**: 1983, 1935.
29. Steiner, Morris, Zuger, Bernard, and Kramer, Benjamin: Production of Renal Calculi in Guinea Pigs by Feeding Them a Diet Deficient in Vitamin A, Arch. Path. **27**: 104, 1939.
30. Higgins, C. C.: New England J. Med. **213**: 1007, 1935; New York State J. Med. **36**: 1620, 1936.
31. Ravdin, I. S., and Frazier, William D.: The Use of Vitamin B_1 in the Preoperative Preparation of the Hyperthyroid Patient, SURGERY **4**: 680, 1938.

ACUTE PERFORATED DUODENAL DIVERTICULUM

A Case Report

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DIVERTICULA of the duodenum have been reported since 1710[1] but their frequency has only been recognized since the recent development of better x-ray technique. The incidence of duodenal diverticula has been reported to be as high as 1.2 per cent.

Diverticula of the duodenum occur more frequently in the latter decades of life. Diagnosis is difficult. The symptoms are usually suggestive of chronic appendicitis, peptic ulcer, gall-bladder disease, or colitis. There is nothing typical of the symptoms. Even when x-ray findings are positive, the gastrointestinal complaints are not necessarily caused by the diverticulum.

Diverticula may be found on all parts of the duodenal wall, but the usual site is on the descending part and in the region of the ampulla of Vater. Usually the diverticulum springs from the inner and posterior aspect of the duodenum along the line of entrance of the vessels. The mucosa and muscularis mucosa are protruded through the muscular wall. Thus, the diverticulum is very thin and it would seem that perforation would be common. However, as a matter of fact, I was able to find only a very few instances of acute perforation of a duodenal diverticulum reported in the literature. Hahn[2] reported two cases with recovery. Monsarrot[3] had one patient who also recovered. Thorek[4] had a case associated with duodenal ulcer and Lucinian[4] reported a remarkable case of a perforated duodenal diverticulum which was diagnosed by x-ray.

Due to the rarity of this disease and the difficulty of making an operative or preoperative diagnosis, I think the following case worthy of report.

Case Report.—A white male, aged 59 years, entered the hospital with the following history: He had awakened at his regular time and gone about the usual activities of farming. After a hearty luncheon, he was resting on the porch when, at about 2 P.M., he was suddenly seized with an intense upper abdominal pain which was so severe that he broke out in a profuse perspiration. He was assisted to a bed and his wife tried to relieve him by the administration of local applications, emetics, and enemas. Finally he was persuaded to send for a physician who saw him at 6 P.M. The family physician made a diagnosis of a perforated ulcer and advised that he come to the city for an immediate operation. After some deliberation and delay, the patient was persuaded to come to the hospital, which he entered at 1 A.M. He stated that he had never been sick before. He denied ever having had indigestion, gas, or any other digestive disturbance. His family history and marital history were irrelevant.

On examination he was found to be a well-nourished white male weighing 175 pounds and having a height of six feet. His temperature was 98.6; pulse, 90;

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respiration, 22; and blood pressure, 120/78. The remainder of the physical examination was normal, with the exception of the findings in the abdomen.

The upper half of the abdomen did not move with respiration. There was very little tympanites and no distention. The upper half was boardlike, but the lower half was quite soft. Except in the epigastrium there was very little tenderness. On auscultation the abdomen was quiet.

The urine was normal. The hemoglobin was 95 per cent; there were 5,000,000 reds and 8,500 white cells with 86 per cent polymorphonuclears and 14 per cent lymphocytes. The shift was to the left.

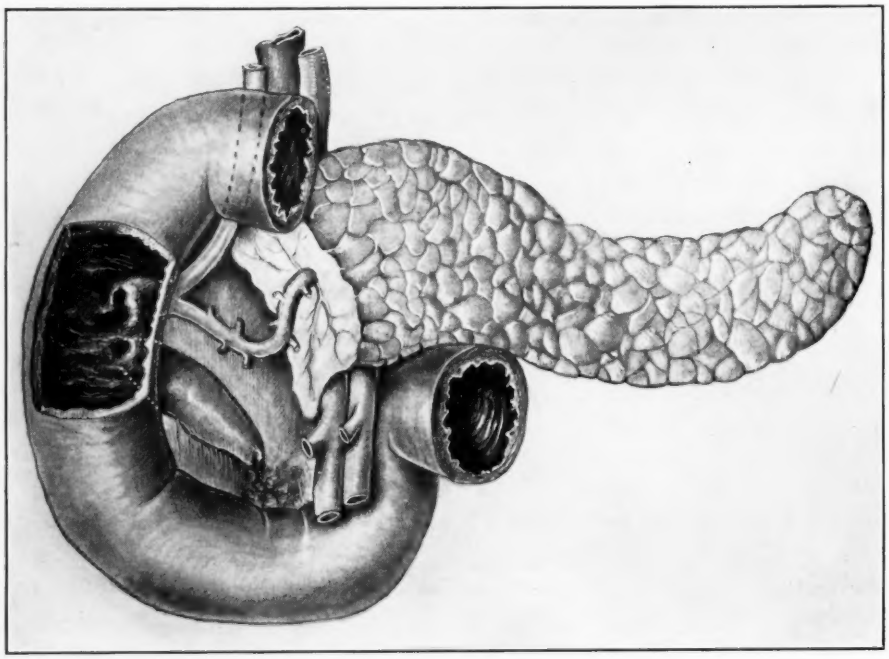

Fig. 1.—A diagrammatic sketch showing a perforated duodenal diverticulum. The head of the pancreas was omitted from drawing and a small window was made in the anterior wall of the duodenum to demonstrate the diverticulum and its perforation at the tip.

The diagnosis of perforated peptic ulcer was concurred in and the patient was prepared for an immediate laparotomy. Under ether anesthesia, which was preceded by an infusion, the patient received an upper right rectus incision. The rigidity persisted to some extent with the patient fully under the anesthetic. The peritoneum was opened, but no escape of gas was noted. About 300 c.c. of a creamy exudate was aspirated from Morrison's pouch and a search started for the perforation. The entire duodenum was apparently free of ulceration. Some of this exudate adhered to the second portion of the duodenum. The stomach was normal. The appendix was found to be acutely inflamed and was removed. There was no perforation in this organ. The remainder of the intestines were injected, but no perforations were found. The pancreas and spleen were normal. The gall bladder was tense and did not empty. There were rather dense adhesions between the stomach and gall bladder. The adhesions were freed and a cholecystostomy performed in the hope that possibly the gall bladder would account for the large amount of exudate. There was no pus in the bile obtained from the aspiration of the gall bladder. The abdomen was closed with the fear that the correct diagnosis had not been discovered.

The patient left the operating room with a blood pressure of 120/80 and a full and regular pulse of 96. Two hours later symptoms of shock appeared, his blood pressure dropping to 80/50 and the pulse developing a rate of 140. Supportive treatment was begun but the patient died within another hour.

At autopsy nothing of importance was found, except around the duodenum. There were small collections of a creamy exudate along the anterior surface of the duodenum. No ulcers were palpated. The peritoneum was stripped off and a large collection of pus and duodenal contents (about 100 c.c.) was found along the second portion of the duodenum. The fluid lay between the duodenum and the head of the pancreas and in front of the right kidney. The duodenum was pulled out of its bed and a diverticulum about the size and shape of the index finger (2½ inches long and ½ inch in diameter) was found in this collection of creamy fluid. The duodenum was split and the opening of the diverticulum was discovered ¼ inch below the entrance of the common duct. This is the usual site for the openings of duodenal diverticula. There was a match-stick perforation at the tip of the diverticulum which was very thin and did not contain a muscular layer.

SUMMARY

A case of a perforated duodenal diverticulum is presented. The symptoms and signs are similar to those of a perforated peptic ulcer. The diagnosis may be missed, as it was in this patient, unless the possibility of such a condition is kept in mind. I would suggest that if the signs and symptoms point to a perforated ulcer and the intraperitoneal pathology reveals nothing more than an exudate along the anterior duodenal wall a small incision be made in the posterior peritoneum and a search begun for a diverticulum.

REFERENCES

1. Boyd, William: Surgical Pathology, Philadelphia, 1933, W. B. Saunders Company, p. 269.
2. Hahn, O.: The Duodenal Diverticulum, Ergebn. d. Chir. u. Orthop. **23**: 392, 1930.
3. Monsarrot, K. W.: Acute Perforation of a Duodenal Diverticulum, Brit. J. Surg. **14**: 179-180, 1926.
4. Thorek, Max: Duodenal Diverticulum With Ulcer, Illinois M. J. **55**: 64, 1929.

PRIMARY ACUTE EPIPLOITIS

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THE TERM epiploon is applied not only to the great omentum, but also to the gastrocolic omentum, the gastrohepatic omentum, and the numerous epiploic appendages found on the large intestines. Acute epiploitis is primary when there is no known etiology. When the inflammatory process is the result of the epiploon's being caught in a hernial sac, or involved in the adhesions of a previous operation or adjacent diseased organ, it is considered secondary.

Eitel,[1] in 1899, was probably the first to describe a case of primary acute epiploitis. His case involved the great omentum and was reported as a rare tumor of the omentum. Baldwin's[3] report of a case in 1902 is said[4] to be the first reported in this country. In more recent years there have been numerous case reports either under the term acute epiploitis or primary torsion of the omentum.

INCIDENCE

Cowell,[5] in a review of the subject in 1925, found only 18 cases of primary torsion of the omentum. Barsky and Schwartz[6] were able to find only 62 cases in the literature prior to 1937. It was therefore thought that it would be worth while to report and discuss 13 cases of primary acute epiploitis.

Eleven of these 13 patients were operated upon in the University Hospital during the fifteen years from 1922 to 1937. During that period there were approximately 30,000 patients admitted to the general surgical service. It would seem, therefore, that the disease is probably more common than one would suspect from a review of the literature. The other 2 cases included in this report were seen by the senior author in other hospitals. Eight of the 13 patients were males. The age varied from 20 to 62 years. There would seem to be no reason that the condition should be confined to any sex or age group. It is notable, however, that there are no children in this series. It has occurred in a child 3 years of age.[16]

ETIOLOGY

Theoretically, primary acute epiploitis could result from (a) strangulation by torsion of the epiploon, (b) interference with the blood supply by embolus or thrombosis, or (c) bacterial invasion of the epiploon through the circulation or from the adjacent intestinal tract. The first of these possibilities was found to be the causative factor in all of the

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cases involving the omentum herein reported. Torsion could be demonstrated in only 1 of the 3 cases involving epiploic appendages. In the other 2 cases the etiology was not known. The epiploon had the appearance of being involved in an inflammatory process, but it could not be demonstrated to have come from the adjacent colon.

The etiology of the torsion is unknown. In one of our cases the pain started suddenly, as the patient turned over in bed. Numerous etiologic possibilities have been suggested in the literature:

1. There is sudden rotation of the body or violent exertion.[5, 6] Only one of our patients gave such a history.

2. There is often a congenital malformation of the omentum[8, 9] so that the part involved is long and narrow and therefore easily twisted on itself.

3. The anatomic arrangement of the vessels may be such that the long tortuous veins twist around the short arteries as the omentum swings from the transverse colon.[6]

4. Venous thrombosis may be the first lesion.[11] The veins would then become distended and would easily rotate around the arteries. Thrombosis of the vessels is always found at operation, but it cannot be demonstrated that it was present before the torsion.

5. An inflammatory process starts first[7] and the torsion follows as the heavy inflamed omentum swings from the unaffected area.

6. The omentum, heavy with fat, is a good subject for torsion, as it swings from the transverse colon. Farr and Bachman[11] have suggested that torsion is more apt to occur in well-nourished individuals. This has been our experience.

PATHOLOGY

The cases involving the omentum herein reported are all samples of primary torsions resulting in acute epiploitis. It seems likely, therefore, that the torsion is the original incident and that thrombosis and strangulation follow this. The torsion has been found to be both clockwise and counterclockwise. There is the usual picture of strangulation with damage to the blood vessel walls and extravasation of blood into the tissues. When the area involved is great, the abdomen usually contains free bloody fluid. Infection is usually not present except as an invader to a point of lowered resistance. In none of our cases was there suppuration present. At operation, a bluish black mass is found, covered by the surrounding structures which are adherent to it by a plastic exudate. In an older lesion the involved omentum becomes completely gangrenous. If left alone, this either would be absorbed[12] or would result in an abscess,[13] depending upon the size of the piece of omentum involved.

In two of the cases involving an epiploitic appendage, torsion was not demonstrated at operation. In the earliest case there was an inflamma-

tory reaction in the fatty tissue of the epiploic appendage, so that it was red and injected and covered with a whitish exudate. In the case of longer duration, the epiploic appendage had assumed a bluish black color and was completely surrounded with omentum.

In the cases in this series there were found three varieties of primary acute epiploitis from the clinical standpoint, that involving (1) the entire greater omentum, (2) a small part of the omentum, or (3) one of the epiploic appendages. Of the cases herein reported, two fall in the first group, three in the third group, and the remainder in the second group.

SYMPTOMS

Abdominal pain was the only constant symptom found in these cases of primary acute epiploitis. The symptoms were not diagnostic and differed in no respect from those frequently seen in acute appendicitis. In 5 instances the pain was generalized and later localized. It is significant, however, that in 7 of the 13 cases the pain was localized from the start and remained so.

Nausea and vomiting were uncommon. Only 4 of the 13 patients vomited. Mullen[14] has reported that vomiting frequently preceded the pain in torsion of the omentum, but this was not the case in this series.

The severity of the pain undoubtedly depends largely upon the size of the strangulating epiploon. In the patients with the entire omentum involved, the pain was severe. One patient was operated upon within five hours of the onset of pain. On the other hand, when a small portion of the omentum or an epiploic appendage is involved, the pain is not severe. Thus these patients reach the surgeon as a rule after a considerable delay. One patient in this group went seven days, and the average was about three days.

FINDINGS

The physical findings depend upon the extent of the pathology. Tenderness was present in all cases. There was some rigidity in all but 3 patients. Of the 13 patients, 6 had a palpable mass. Several others were so rigid that the mass could not be palpated. The findings were on the right side of the abdomen in 10 of the 13 cases. In 6 instances, the findings were definitely in the right lower quadrant.

The blood count was helpful only in indicating the presence of inflammation. The W.B.C. was under 10,000 in only 1 instance. The highest was 16,500.

The patients had very little fever, as a group. The highest temperature upon admission to the hospital was 100.3°. The temperature was below 99° in 7 of the cases reported.

DIAGNOSIS

The diagnosis was made preoperatively in only 2 of the cases. These were both on the left side and had a small tender mass palpable. The

usual diagnosis in these cases is acute appendicitis. This was true in 8 of this series. When a mass was palpable, it was thought to be an appendical abscess. In 2 instances, where the pathology was in the right upper quadrant, the diagnosis was acute cholecystitis. When a large portion of the omentum is involved and the patient is seen early, the findings may simulate those of a ruptured ulcer.[10] A mass of twisted omentum in the flank has simulated kidney pathology.[15]

The effort to make the diagnosis of primary acute epiploitis preoperatively is a matter of academic interest. It is true that one may often suspect the diagnosis, but it is impossible to be certain. It is sufficient to make the diagnosis of an acute surgical abdomen and to localize the pathology. To attempt to diagnose a strangulation of a small bit of omentum and thus avoid operation is a hazardous procedure. In this respect, it may be similar to the diagnosis of acute mesenteric lymphadenitis in children. The diagnosis is often made and proved to be correct at operation, but one cannot be certain enough to avoid operating for appendicitis. To depend upon a nonoperative diagnosis of this sort sooner or later will lead to trouble from a ruptured appendix.

TREATMENT

The treatment of primary acute epiploitis is resection of the involved epiploon. The diseased tissue is usually surrounded by and adherent to the adjacent structures by a plastic exudate, except in the early cases. It is easily separated from the surrounding structures by finger dissection.

In 2 instances in this series, a resection of the omentum was not done. In one case the pain stopped suddenly before operation and the omentum was found at operation to have become untwisted. In a second case the omentum was untwisted at operation and was obviously viable. This latter procedure is advised against because of the danger of an embolus[17] from thrombosed vessels. Thus, the omentum should not be untwisted unless it can be demonstrated that even thrombosis is not as yet present.

It is undoubtedly true that when only a small segment of omentum is involved, spontaneous recovery may occur by absorption of the necrotic material. It is also probable that if absorption does not occur and an abscess develops, it will be well walled-off. For this reason, no doubt, Schrager and Bergen[18] have advocated nonoperative treatment at first, with late drainage of the abscess should it occur. This we consider poor surgical practice, especially since a positive diagnosis cannot be made before operation.

RESULTS

The results of surgical excision of the inflamed epiploon are uniformly good. These patients run the usual risks of the complications

TABLE I

AGE	DURATION OF SYMPTOMS IN DAYS	PAIN LOCALIZED FROM START	VOMITING	TENDERNESS	RIGIDITY	PALPABLE MASS	LOCATION	W. B. C.	PREOPERATIVE DIAGNOSIS	OPERATIVE FINDINGS
47	7	x		x	x		RUQ*	12,300	Acute gall bladder	Inflamed epiploic appendage
62	¼			x			Right of umbilicus		Acute appendicitis	Entire omentum recently untwisted
20	5			x	x	x	RLQ	14,500	Appendical abscess	Torsion right half omentum; free bloody fluid
47	4		x	x	x		RLQ	11,000	Acute appendicitis	Torsion right half omentum; free bloody fluid
40	2	x		x	x	x	RUQ		Acute gall bladder	Torsion part of omentum; gangrenous
27	3			x	x	x	Left of umbilicus	14,800	Acute epiploitis	Torsion part of gastrocolic omentum
37	1	x		x	x		RLQ	12,000	Acute appendicitis	Torsion small part of omentum
41	2	x	x	x	x	x	RLQ	16,500	Appendical abscess	Torsion right half of omentum
30	1			x			RLQ	16,500	Acute appendicitis	Torsion small part of omentum
20	1	x		x			Right of umbilicus	9,300	Acute appendicitis	Torsion small part of omentum
23	2	x	x	x	x		RLQ	14,800	Acute appendicitis	Torsion entire omentum; bloody fluid
38	4	x		x	x	x	LLQ		Acute epiploitis	Torsion of epiploic appendage
40	3		x	x	x	x	LLQ		Diverticulitis	Inflamed epiploic appendage

**RUQ*, right upper quadrant; *RLQ*, right lower quadrant; *LLQ*, lower left quadrant.

and catastrophes of surgery naturally. One patient in this series died of postoperative pneumonia. The abdomen was clean at autopsy. In none of the patients in this series had the pathology progressed to abscess formation. In such an event the prognosis should be good with surgical drainage, and the course of events similar to that seen in an appendical abscess.

The patients in this series have been followed from three months to fifteen years and in no instance has there been a recurrence of the trouble.

SUMMARY

1. Primary acute epiploitis is an uncommon condition of which thirteen cases are herein reported.

2. The diagnosis of primary acute epiploitis should be made, only to be confirmed at operation.

3. The treatment is excision of the inflamed epiploon.

REFERENCES

1. Eitel, G. G.: Quoted by D'Errico.[2]
2. D'Errico, E.: Primary Torsion of the Great Omentum, New England M. J. **203**: 1181-1188, 1930.
3. Baldwin, J. F.: A Contribution to the Study of Intra-Abdominal Omental Torsion, Ann. Surg. **36**: 940, 1902.
4. Wallace, R. H., and Miller, R. H.: Primary Omental Torsion, New England M. J. **208**: 831-932, 1933.
5. Cowell, E.: Abdominal Torsion of the Omentum, Brit. J. Surg. **12**: 738-751, 1925.
6. Barsky, E. K., and Schwartz, A. M.: Primary Omental Torsion. Review of Literature and Report of Two Cases, Am. J. Surg. **38**: 356-363, 1937.
7. Jeffries, J. W.: Torsion of the Omentum, Ann. Surg. **93**: 761-765, 1931.
8. McWhorter, G. L.: Torsion of the Omentum Without Hernia, Arch. Surg. **16**: 569-582, 1928.
9. O'Brien, G. G.: Congenital Anomaly of the Omentum Causing Torsion, J. A. M. A. **102**: 681-683, 1934.
10. Smyth, M. J.: Abdominal Torsion of the Omentum, Lancet **2**: 572-576, 1930.
11. Farr, C. E., and Bachman, R. F.: Torsion of the Omentum, Ann. Surg. **97**: 766-772, 1933.
12. Galloway, W. D.: Primary Abdominal Torsion of the Omentum With an Attempt at Spontaneous Cure, Brit. M. J. **2**: 899, 1935.
13. Thorek, M., and Thorek, P.: Post-Operative Epiploitis With Abscess, J. A. M. A. **98**: 1546-1549, 1932.
14. Mullen, T. F.: Torsion of the Great Omentum, Surg., Gynec. & Obst. **40**: 635-641, 1925.
15. Salsbury, C. R.: Omental Torsion With Unusual Symptoms, Brit. J. Surg. **23**: 115-118, 1935.
16. Morris, J. H.: Torsion of the Omentum, Arch. Surg. **24**: 40-76, 1932.
17. Robb, D.: Abdominal Torsion of the Omentum, Lancet **2**: 1347, 1930.
18. Schrager, V. L., and Bergen, S.: Primary Acute Epiploitis, Ann. Surg. **20**: 45-50, 1933.

ISOLATED INJURY OF THE MESENTERY WITHOUT PERFORATION OF THE ABDOMINAL WALL

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ISOLATED injury of the mesentery without perforation of the abdominal wall is so rare that it seems worth while to report such a case, if only so that it may be on record.

In 1920 Prutz[1] could collect only 36 cases. Eighteen of these were operated upon, 8 dying; in 18 not operated upon, all died.

By 1936 Mandillon and Poinot[2] were able to review 70 operated cases and added 3 of their own. From a mortality of 57 per cent they conclude that the prognosis depends mainly upon the time elapsing before operation, the extent of the injury, and the degree of shock.

Case Report.—Jackson Memorial Hospital No. C-7097. On Sept. 7, 1937, at about 6:00 P.M., a 2-year-old white girl was playing beside a 4 foot wall built of 4 by 8 inch concrete blocks between which the cement had not hardened. Each block weighed approximately ten pounds. When a 12-year-old girl leaned over this wall to pick up the child, the wall collapsed and the baby fell beneath girl and blocks. Vomiting occurred at once and in about fifteen minutes there was an involuntary stool. The baby retained consciousness and shortly began to cry. She was given a hypodermic (morphine?) by a physician and promptly went to sleep. About 1:00 A.M. she awakened screaming so was taken to the hospital.

Physical examination at 2:00 A.M. revealed a normally developed and well-nourished white female of about 2 years of age, who presented a picture of acute shock: thirst, marked pallor, great restlessness; pulse, 160 plus, very weak; respirations, 32 to 40, shallow and grunting; profuse sweating. The abdomen showed several small contusions over the upper half and was tense and did not move with respiration; definite muscle spasm approaching rigidity, especially in upper left quadrant; extreme tenderness throughout, but most marked in upper left quadrant; no peristaltic sounds heard; shifting dullness in the flanks. Rectal examination revealed fullness and tenseness in the cul-de-sac. Heart and lungs presented no abnormalities. Blood pressure, 88/50; voided urine, negative; R.B.C., 4,150,000; hemoglobin, 71 per cent; W. B. C., 27,700; polymorphonuclears, 86 per cent. Upright x-ray of the abdomen showed an intact diaphragm and no subdiaphragmatic gas.

Diagnosis.—Acute shock, intra-abdominal hemorrhage, most probably from ruptured spleen. Antishock treatment, including a transfusion of 200 c.c. of citrated blood, so relieved the condition of acute shock that it was felt the baby could stand operation.

Operation.—At 4:00 A.M., under drip ether anesthesia, the abdomen was opened through an upper left rectus incision. A small hemorrhagic area was encountered in the muscle, apparently ecchymosis from the trauma. On opening the peritoneum, free bright blood escaped, suction recovering 200 c.c., besides a quantity

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of clots eventually sponged out. The spleen, liver, diaphragm, and anterior wall of stomach were uninjured. There was no free blood in the lesser sac. The omentum, colon, and mesocolon were intact. Exploration of the small intestine and its mesentery revealed a laceration in the mesentery through both leaves at approximately its midportion. This laceration was about 15 cm. in length, beginning 1 cm. from the posterior attachment and extending to 1 cm. from the intestine. There were many freely bleeding points, all apparently venous, as no spouting points were observed and the pulsations of an artery could be seen in the edge of the rent. The adjacent intestine was healthy looking with moist, glistening serosa so it was felt that its circulation was not impaired. All bleeding points were tied and the laceration repaired by opposing the peritoneal edges on both sides with interrupted sutures, no through-and-through sutures being placed. The abdomen was closed in layers without drainage.

The postoperative course, though a bit stormy at first, caused no real concern at any time and the baby was discharged on Sept. 18, 1937. She has been seen at frequent intervals and never have any ill effects of the injury been noted. Now, fourteen months later, she is not constipated and is normal for her age in every way.

COMMENT

This case presented an interesting diagnostic problem, both preoperatively and at operation. The picture was certainly one of intra-abdominal hemorrhage. With hollow viscera and kidneys apparently ruled out, rupture of liver or spleen seemed most likely. Location of area of greatest tenderness and of greatest spasticity in left upper quadrant pointed most logically to the spleen.

The described injury might easily be overlooked in the event of first finding other injury; this could account for otherwise unexplained deaths following trauma to the abdomen.

If unoperated recovery from such injury should occur, the circulation to a loop of intestine might be so impaired as to cause a stenosis,[3] possibly so long afterwards that history of trauma could not be elicited.

Operation as soon as the condition of the patient warrants it is the undoubted treatment, and, in view of the reported 57 per cent mortality, is obviously often delayed too long.

REFERENCES

1. Stocker-Dreyer: Subacute Isolated Injury of the Mesentery, Schweiz. med. Wchnschr. **50**: 90, 1920.
2. Mandillon, G., and Poinot, J.: Abdominal Contusions With Multiple Lesions of the Mesenteric-Intestinal Junction, Rev. de chir., Paris **55**: 578, 1936.
3. Pupini, Guido: A Case of Post-Traumatic Stenosis of the Small Intestine, Policlinico (sez. prat.) **39**: 847-852, 1932.

RHABDOMYOSARCOMA

Report of Case Presenting Unusual Features With Reference to Age, Race, Site of Origin, and Manner of Growth

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RHABDOMYOMATOUS tumors are usually found in young persons and children in such positions as the heart wall, bladder, vagina, kidney, esophagus, and uterus. These tumors contain cells of many varieties and among them are found atypical, striated muscle fibers. However, it is unusual to find a tumor composed exclusively of muscle tissue and the admixture is often with such constituents as cartilage, loose cellular connective tissue, myxomatous areas, and even epithelial structures. The suspicion, therefore, is aroused that the rhabdomyomas are closely related to the mixed tumors or teratomas.[1] Rhabdomyomas of the skeletal muscles are rare and of the 18 cases reported up to 1913, Kuttner[2] rejected 6 and reported the others as questionable. A search of the tumor registry of the Army Medical Museum by one of us (J. M. F.) revealed several authentic cases in which the neoplasm originated in a skeletal muscle. None of these, however, arose from the sternocleidomastoid muscle which was the location of the tumor reported here.

Case Report.—F. D., male, colored, aged about 75 years, entered the hospital on Oct. 18, 1938, complaining of a large tumor mass on left side of neck. The patient did not appear to be acutely ill and his chief difficulties arose from inability to rotate his head and from respiratory embarrassment due to pressure on the trachea by the tumor.

Family History.—Father had "lumps on neck for years that Doctors called scrofula." This patient, however, died from an attack of typhoid fever at the age of 66 years. The family history otherwise was irrevelant.

History of Present Illness.—The patient had enjoyed excellent health all of his life. He stated that in the region of the present neoplasm, there had existed a small nodule about the size of a hen's egg for about fifty years. About four months ago this had begun to enlarge without accompanying pain and remained symptomless until its bulk mechanically interfered with head rotation and respiration. No history of trauma preceding this sudden activation of the tumor could be elicited.

Physical Examination.—There was a tumor mass about the size of a grapefruit lying to the left of the midline of the neck (Fig. 1). The overlying skin was smooth and shiny. The neoplasm beneath was densely adherent to the skin and to surrounding structures. The mass was hard and nodular, irregular in outline, and had no signs of fluctuation.

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The borders of the tumor were roughly mapped out to be the ramus of the mandible above, the sternoclavicular angle below, the posterior auricular space posteriorly, and anteriorly the anterior belly of the right sternocleidomastoid muscle.

Aside from a mild systolic murmur and arteriosclerosis, the remainder of the physical examination was essentially negative.

Radiologist's Report.—Nov. 5, 1938 (Dr. Frank Wrenn), x-ray of chest showed: The right lung was apparently clear throughout. The left lung showed a consolidated area in the midportion. This was not sharply marginated, but was strongly suggestive of a metastatic lesion.

Operation.—The mass was exposed by a longitudinal incision under local anesthesia. There was no suggestion of encapsulation and no line of cleavage could be determined; therefore, it was necessary to dissect the tumor free from the surrounding structures. As vascularity was quite marked, the amount of bleeding caused by this procedure was considerable.

Fig. 1.

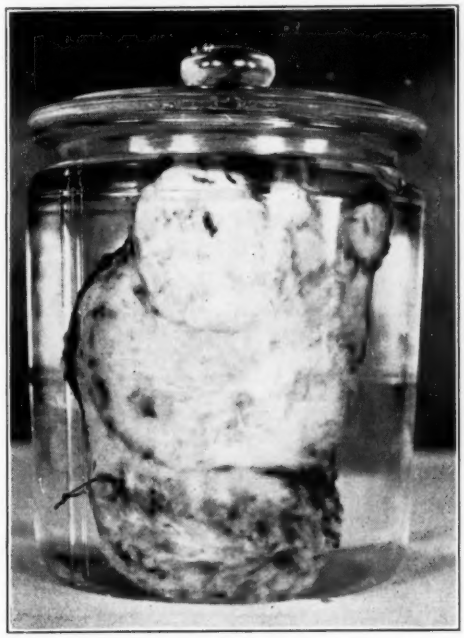

Fig. 2.

Fig. 1.—Tumor mass in neck. Nodular characteristics apparent in this photograph.

Fig. 2.—Excised specimen. Note three distinct zones. The upper contains the calcified nodules; the middle is dense and shows cartilage-like material interspersed with rosettes of soft, pink tissue. The lower zone is composed of very soft, friable tissue, so necrotic that some of it washed away in the fixing solution.

Gross Examination of Tumor.—The excised mass weighed 1,100 gm. and measured 17 by 11 cm. It was nodular, dusky red in color, and the outer surface contained a number of tabs of connective and normal-appearing muscle tissue. The cut surface was bathed in glairy mucus and showed a wide variation in structural detail. (Fig. 2.) It was apparent that three distinct zones could be classified. The upper contained several nodules of bony hardness which required the use of a saw to execute their division.

In the central zone the tissue was of a soft cartilaginous texture, yellowish in color. Into this were inset several pink rosettes of muscle-like substance, the radiating arms of which finally blended and were lost in the cartilage frame. In

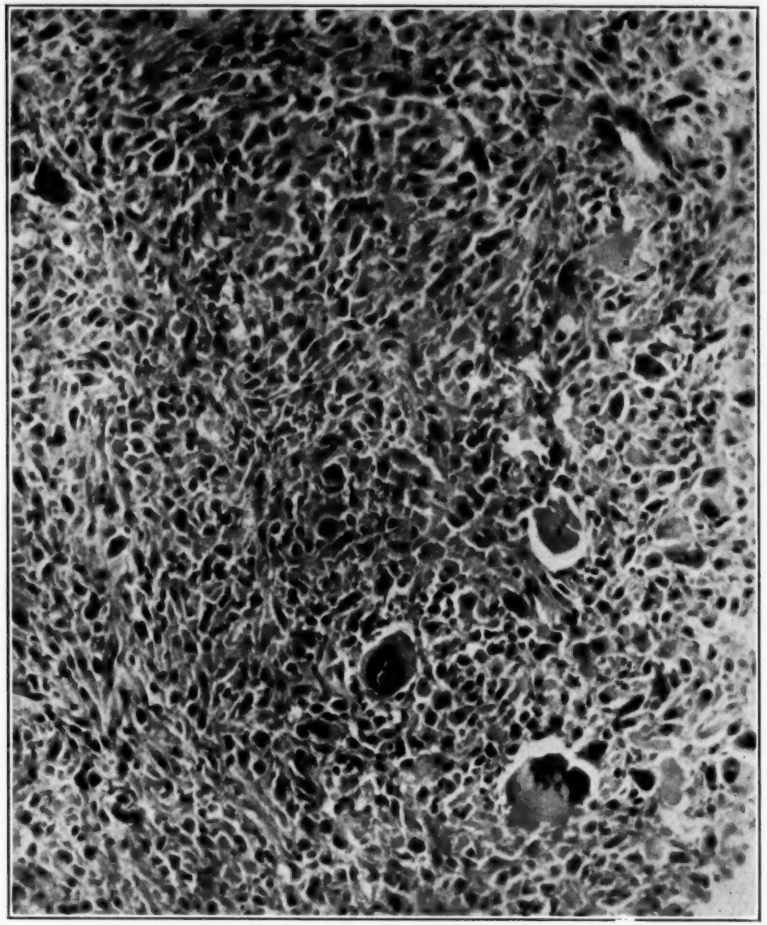

Fig. 3.

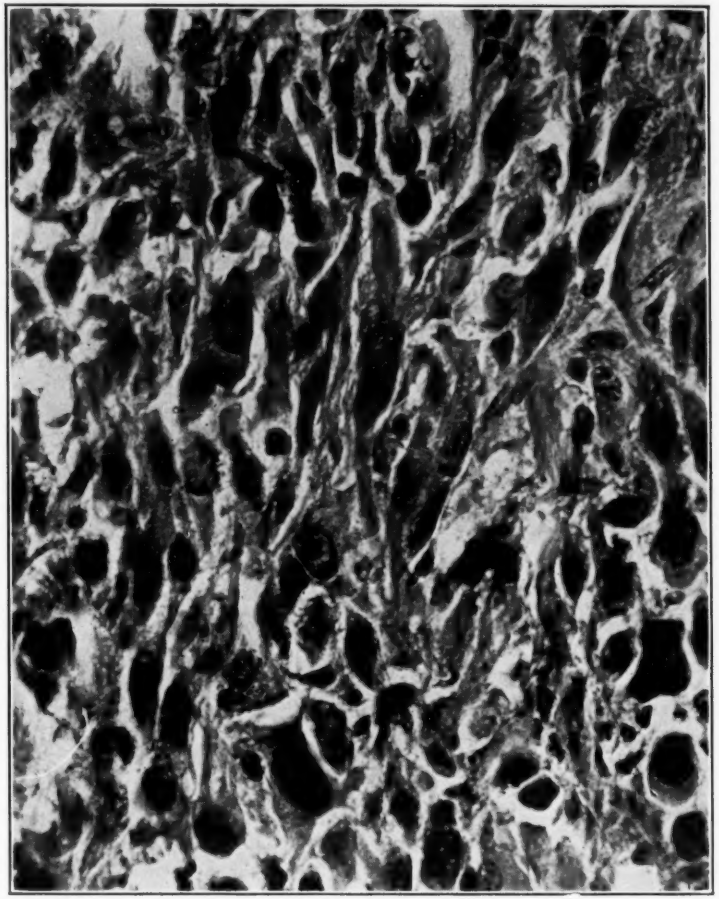

Fig. 4.

Fig. 3.—Photomicrograph showing irregular arrangement of fusiform, acidophile cells, with liberal sprinkling of bizarre giant cells.

Fig. 4.—High magnification photomicrograph, showing striation and vacuolization of nuclei. In the lower central area a typical "spider cell" can be seen. Due to reproduction of picture, some of the details have been diminished.

the lower zone the tissue was very soft and friable, so much so, in fact, that portions of it washed free in the fixing solution.

Microscopic Examination.—Sections cut from the bony hard nodules of the superior pole failed to show evidence of ossification. Only hyalinization was demonstrated. Blocks cut from the cellular areas varied somewhat in their general architecture, but the picture had an essential sameness, showing irregularly arranged strands and masses of acidophile, fusiform cells separated by a scant, hyalinized stroma. (Fig. 3.) In many instances longitudinal striation was marked and in a few cases transverse striation could be demonstrated. Liberally sprinkled among these fusiform cells were bizarre giant cells, which were more plentiful in some areas than in others. Some of these giant cells showed striation and vacuolization of nucleus, giving them all of the characteristics of the "spider cell" described by Ewing.[3] (Fig. 4.)

Diagnosis.—Rhabdomyosarcoma. (Sections were sent to Drs. James Ewing, A. C. Broders, Edgar A. Pund, Kenneth Lynch, and J. E. Ash, with the request that an opinion be given. In each instance these authorities concurred with the diagnosis made by us.)

Postoperative Progress.—In the six weeks that have elapsed since operation the patient's course has been steadily downhill. He has grown gradually weaker until he remains in bed all of the time. Sleeplessness has to be combatted by use of strong sedatives. Cough is very bothersome and considerable mucus is expelled. The site of operation is densely edematous and it is believed that there is a beginning local recurrence of the tumor.

Radiologist's Report.—Dec. 20, 1938 (Dr. Frank Wrenn): In the right lung, just above the diaphragm, there is a round, opaque shadow about 2 cm. in diameter. In the left lung there are several round, opaque shadows throughout the lower lobe, varying from 0.5 to 3 cm. in diameter. In the upper lobe there is one similar shadow about 2 cm. in diameter. These shadows are interpreted as being metastatic lesions from a new growth. The development has been very rapid since the report of Nov. 5, 1938.

COMMENT

A rhabdomyosarcoma is reported which originated in the left sternocliedomastoid muscle of a colored man 75 years of age. We can find no record of a similar tumor arising in this site; likewise, no record can be found of this neoplasm appearing in the skeletal muscles in the colored race.

The age of the patient presents a most unusual aspect, and the fact that the tumor lay dormant for more than fifty years and then suddenly sprang forth in full malignancy appears to lend support to the theory that rhabdomyomas are closely related to the mixed tumors or teratomas.

We wish at this time to express our gratitude to the several eminent authorities who so graciously aided in confirming the diagnosis. We are also indebted to the courtesy of Lieut. Col. J. E. Ash, Medical Corps, U. S. Army, for supplying photomicrographs and to Dr. Frank Wrenn and Miss Fronde Stewart, of the Department of Radiology of the Anderson County Hospital, for their gracious aid in x-ray studies and in preparing photographs used in illustrating this article.

REFERENCES

1. MacCallum, W. G.: Text Book of Pathology, Philadelphia, 1928, W. B. Saunders Company, page 962.
2. Kuttner, A.: Deutsche. chir. **25**: 1913.
3. Ewing, James: Neoplastic Diseases, ed. 3, Philadelphia, 1931, W. B. Saunders Co., page 235.

VENOUS MESENTERIC OCCLUSION

A Clinical Discussion and Experimental Study

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MESENTERIC vascular occlusion and mesenteric thrombosis are terms which, as used clinically, describe those disorders in which an obstruction of the mesenteric vessels is present, but in which a direct occlusion of the lumen of the gut itself is not associated. In several conditions, as in volvulus or intussusception, the direct occlusion of the mesenteric vessels which may accompany obstruction of the gut lumen may cause a more fulminating picture than gut lumen obstruction itself. But by clinical custom, the mechanical features referring to gut lumen obstruction dominate the diagnosis. In other words, pathologically speaking, mesenteric vascular occlusion or mesenteric thrombosis occurs sooner or later in several conditions ordinarily listed under the heading of intestinal obstruction; but in the clinical world we have come to associate the terms predominantly with a particular syndrome. This syndrome for our purpose may be described aptly by a quotation from one of our popular textbooks: "Symptoms are sudden, violent, diffuse abdominal pain with collapse, sub-normal temperature, and signs of ileus.[1] There is leukocytosis, a late temperature, palpable mass, evidence of free fluid, and early peritonitis." Further quotations from medical and surgical textbooks could be cited to illustrate that this fulminating picture is the one usually associated with the terms mesenteric occlusion or mesenteric thrombosis. However, there is some textbook mention of a more slowly progressing occlusion. And a number of writers, including Larson, Meyer, Douglas, Ochsner, Cave, Held, and Goldbloom, and one of us have mentioned or discussed in the journals a slower more deceptive type. Nevertheless the clinical conception of mesenteric vascular occlusion is a somewhat inadequate one, frequently not taking into full cognizance the varying manifestations and possibilities which the clinical term implies. Some of the confusion attendant upon the subject is illustrated in the following quotation from Meyer: ". . . . more detailed information is necessary to enable one to determine positively whether or not a segment of bowel will recover function or if changes leading to death from toxemia or gangrene will occur. In Garcia's case there was a grey color, lack of tone, dilatation of the bowel over a large area, yet recovery occurred promptly without resection. In Penninger's case only one foot of bowel appeared anemic and

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reacted sluggishly to stimulation; no resection was done and at post mortem six days later this segment was found to be gangrenous. In one of Davis' cases the entire small bowel seemed gangrenous, yet recovery occurred without resection, while in one of Cabot's cases the color seemed to return but the bowel was found to be gangrenous the next day. Loop had a similar experience.''[6]

In this presentation we will discuss only venous occlusions in which the lumen of the gut is *not* obstructed. When venous obstruction occurs in closed loops, the rapid collection of fluid and gas within the loop secondary to a pure venous occlusion brings about rapid loop distention and undoubtedly rapid secondary interference with arterial supply. Since much experimental work has been done by others with the closed type of loop, we would stress the difference in the pathology of the two types and the fact that we refer here to unobstructed loops.

In 1935 one of us pursued experimental and clinical observations directed at more clearly identifying the slower more deceptive clinical course which may be associated with a pure venous mesenteric occlusion in which the gut lumen remained unobstructed.[2] At this time we discussed the etiology of this condition, and upon the basis of our own clinical and experimental observations as well as some case reports of others, we made the following statement regarding the symptoms of a certain type of venous mesenteric thrombosis: ''Since venous mesenteric thrombosis may occur as a complication of various conditions . . . . it is obvious that the picture may be complicated by various elements of pathology. Generally speaking, however, except in that type of venous mesenteric thrombosis initiated for example by sudden volvulus or intussusception, the onset and course would seem to be relatively slow, and initiated by pains of a moderate colicky nature. Two of our case histories here reported and two other similar histories of cases found in the recent literature, are particularly interesting as regards the several days' duration of symptoms before the victims seem to have given themselves particularly serious consideration. Another striking symptom in the syndrome of this disease is the disproportion of the abdominal tenderness and the duration of symptoms, to the abdominal rigidity. The abdomen is usually widely and definitely tender to deep palpation and the abdominal rigidity is not nearly as marked as one might expect it to be when the possible duration of the complaint and definite tenderness are considered. The normal leucocyte relationship is only moderately disturbed in the early stages, but is markedly distorted later. An engorged intestinal wall may or may not be felt as a palpable mass. Occult blood is uniformly present in the stool and bowel movements continue, dark and soft to liquid in nature. Dark brown 'coffee ground' like vomitus may supervene. The temperature range in the uncomplicated picture is low. Intestinal 'fluid levels' are usually absent. Unless the possibility of the disease is kept in mind it will be invariably

very difficult to recognize it as a definite surgical condition in its earlier stages when surgery should offer a very excellent prognosis. Intestinal obstruction, appendicitis, peritonitis, peptic ulcer, lead colic, 'simple' enteritis, cholecystitis, gastritis, typhoid fever, are among the diseases most likely to be confused by the expert.''

We demonstrated in a small series of experiments accompanying this previous discussion that occlusion of the veins alone in segments of gut up to 25 cm. in length would cause in many instances sufficient edema, discoloration, and sogginess of the gut to indicate that gangrene was present or would supervene; and that, with this condition existing, a recovery of the animal might occur. We showed that in this small series, larger areas of gut could be involved in a pure venous occlusion with recovery of the animal ensuing than was possible when the arteries were involved. We concluded that in rather marked venous occlusions arterial supply at times remained sufficiently unimpaired and the gut wall sufficiently undamaged that a collateral circulation through the omentum and adjacent mesentery in conjunction with lymphatics, eventually would establish sufficient circulation to allow clinical recovery. Further thought upon this subject of venous mesenteric occlusion and comments of others similar to the quotation from Meyer above relative to confusion regarding it stimulated our curiosity and we decided to carry out some additional simple experiments. We concluded previously that, even though some of our experimental animals recovered completely, resection of the gut was, as a rule, probably the safest procedure to follow when venous mesenteric occlusion was recognized.

We are following two clinical objectives in this presentation. One is to attempt to establish more definite clinical concepts of venous mesenteric occlusion and to contrast venous with the more frequently described but less frequently occurring arterial occlusion. The second objective is to attempt a more precise correlation of the pathology of venous occlusion with clinical manifestations, prognostic and therapeutic indications.

EXPERIMENTAL PROCEDURE

The experimental element of this paper has resolved itself in one sense perhaps to determining the extent to which collateral circulation will establish itself in venous occlusions. But one following this subject closely can see that an attempt to surmise at the operating table as to when and when not circulation might become sufficiently established before gangrene or destruction of the gut occurs, might be more difficult than appears on first thought. As most experienced surgeons know, a number of tests directed at determining the viability of the gut at the operating table have been described and used with somewhat questionable success.

As far as we have been able to determine, circulation within the gut of a dog is sufficiently similar to that of the human being for us to

assume that conclusions which have been reached here apply fairly accurately to human beings.

Fig. 1 diagrammatically illustrates the simple technique which we used in producing occlusion of the mesenteric veins. We selected the desired site and length of gut to be involved and placed the mesentery on a stretch with the finger at the sites selected for the ligatures. By rolling the vessels gently on the stretch over the finger, in most instances we were able to insert a needle between the artery and vein and encircle the vein with a small ligature without damage to the artery and with inclusion of very few of the lymphatic radicals. The lumen of the gut was left undisturbed. All the veins in the mesentery leading from the area to be involved were ligated at the sites of easiest election in such a manner that no venous return through the segment of involved mesentery was possible. Though we have on several occasions cut the mesentery along the gut margin between the mesenteric vessel radicals to insure severance of any minute collaterals within the mesentery, we feel that this is in most instances unnecessary. The weblike collaterals seen between the main vessel branches in the mesentery of most dogs are so small and scanty that they are incapable within themselves of producing any worthwhile collateral function in a short length of time.

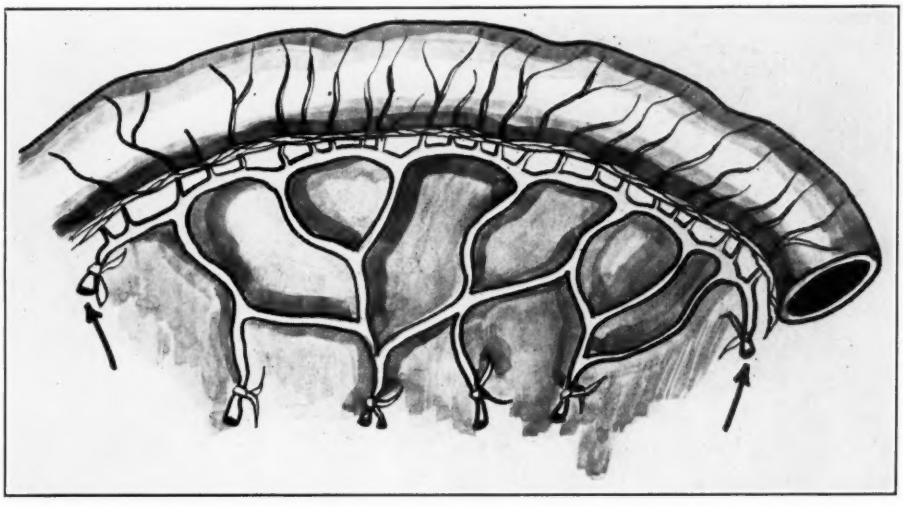

Fig. 1.—Diagrammatic illustration of method used in ligating veins to loops selected. Before loops were returned to abdomen, all were observed after ligations were completed until venous congestion supervened. Arteries and lymphatics are not shown in this diagram for the sake of clarity.

The lower ileum was the site most frequently selected, though the jejunum was sometimes used.

Following completion of the ligation, the potential sources of collateral circulation are few. Those which may be developed from structures which may later adhere to the involved gut, such as omentum, and that circulation which may develop or be carried on through adjacent un-

damaged bowel wall tissues are to be considered. The mesenteric lymphatics also are potential channels for the egress of some edema.

The degree of damage which was consistent or inconsistent with clinical recovery and arbitrary mortality percentages was determined as indicated in the accompanying tables. Other observations, reflecting possibly upon the clinical aspects of this problem, were also made.

Forty-seven animals were used in the study, though nine of these were discarded in the final analysis.

EXPERIMENTAL RESULTS AND DISCUSSION

In comparing the results of venous occlusions with arterial occlusions, it is well to remember that it has been stated that collateral arterial circulation through the tissues of the bowel wall itself can be successfully carried on over a maximum length of about 2 inches. A review of Table I shows the results of sudden venous occlusion without gut lumen obstruction in segments of small bowel 20 to 40 inches in length. In the sixteen dogs in which these lengths were used one dog may be

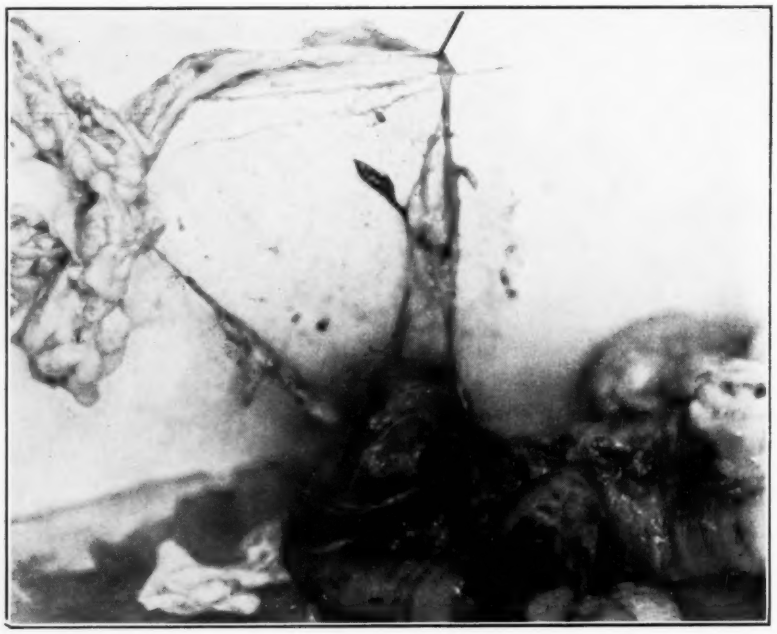

Fig. 2.—This is a photograph of a segment of small bowel which showed recovery following a venous occlusion. Adhesions involving adjacent structures and omentum have been separated. Though it cannot be clearly seen in the photograph, definite vessels are running within the two heavy strands clearly visible in the omentum. These vessels, as may be seen, are closely adherent to the loop and on dissection it could be demonstrated that definite collateral branches from them had established themselves in the gut wall.

said to have recovered. This animal (Dog 2, 20-inch segment) lived sixty-eight days, and, though the bowel was not completely repaired, we believe death was not due to bowel involvement. Dogs 3 and 15 lived twelve days from the onset of occlusion and at autopsy the bowels ap-

TABLE I

VENOUS OCCLUSION LOOPS (GUT LUMEN NOT OBSTRUCTED)
(20 TO 40 INCHES)

DOG NUMBER	SERIAL NUMBER	DATE OPERATED UPON	LENGTH OF LOOP INVOLVED	RESULTS	REMARKS
1	S10-D4	7/24/38	20 in.	Found dead 7/25/38	Gut black, considerable bloody peritoneal fluid; not perforated
2	S10-D7	7/25/38	20 in.	Found dead 10/1/38	Loop involved in Grade 2 adhesions to self and mesentery
3	S10-D11	7/31/38	20 in.	Found dead 8/12/38	Gut loop slightly distended; deep gray-black color
4	S10-D14	7/30/38	20 in.	Found dead 8/2/38	Loop perforated, peritonitis beginning
5	S10-D16	8/ 2/38	20 in.	Found dead 8/3/38	Loop very black; no perforations, peritonitis, or ileus
6	S10-D18	8/ 9/38	20 in.	Found dead 8/10/38	Loop very black; no peritonitis or perforations
7	S10-D28	8/18/38	22 in.	Found dead 8/19/38	Bloody fluid peritoneal cavity; no perforations; loop edematous, heavy, very dark; no peritonitis
8	S10-D15	8/ 2/38	25 in.	Found dead 8/3/38	Loop black; no perforations or peritonitis
9	S10-D23	8/11/38	30 in.	Found dead 8/13/38	Gut deep purple, distended; no perforations
10	S10-D22	8/11/38	30 in.	Found dead 8/12/38	Gut deep purple, distended
11	S10-D21	8/11/38	30 in.	Found dead 8/12/38	Loop almost black
12	S10-D20	8/11/38	30 in.	Found dead 8/12/38	Gut loop deep purple; no perforations
13	S10-D19	8/11/38	30 in.	Found dead 8/11/38	Loop very black; no perforation
14	S10-D17	8/ 9/38	30 in.	Found dead 8/10/38	Loop black; no perforations or peritonitis
15	S10-D13	7/31/38	30 in.	Found dead 8/12/38	Gut deep red in color, somewhat distended adhesions forming; not particularly edematous; no peritoneal fluid; cause of death unknown; dog apparently could have recovered
16	S10-D12	7/31/38	40 in.	Found dead 8/1/38	Loop very dark

Died within 72 hr.	13 (81.2%)
Lived 12 days	2 (12.5%)
Recovered	1 (6.2%)
Mortality	93.7%

peared to be in a condition possibly compatible with recovery. The remainder of animals in this group except two died within 72 hours. The following points are of interest: (1) The first thing of note is that ultimate recovery without surgery must be rare when segments of the small gut of 20 to 40 inches are involved in pure complete venous mesenteric occlusion even with the lumen of the gut unobstructed. Counting Dog 2 as a recovery, the mortality in this group would be 93.8 per cent. Even though this is high, it is not as high as the mortality of arterial

occlusions of this length. The latter give a 100 per cent mortality without surgery. (2) A point of special interest is the relative slowness with which the clinical manifestations occasionally may progress even in these lengthy loops in which sudden venous occlusions have occurred. In two animals (Dogs 3 and 15) it may be seen that twelve days elapsed before death occurred. This is in marked contrast to the rapid (death in twenty-four to forty-eight hours) declination which invariably follows all complete arterial occlusions of the same lengths, and is in keeping with a type of syndrome of venous mesenteric occlusion which has been described as clinically possible in human beings when even rapid or sudden venous occlusions apparently have occurred.

In Table II (segments 12 to 16 inches in length) it is seen that one dog lived sixteen days following the occlusion before dying, substantiating again the clinically slow syndrome which may occur in venous occlusions. One animal (Dog 4) died from intestinal obstruction from adhesions about the involved site; and we are disposed to count the bowel wall in this animal and in Dog 9 as compatible with potential clinical recovery. Doing this, the percentage of death is calculated as 81.8 per cent. In the remaining animals of this group, death occurred within three days.

In the eleven dogs (Table III) in which we used 10-inch segments (25 cm.) the mortality rate diminished but remained quite high (72.7 per cent). Again here it may be seen, however, that the slowness of the syndrome in these relatively short occlusions may be rather pronounced (though as seen from the tables it is frequently fulminating) and that experience with it explains the clinical manifestations and slowness of the progress that has been observed in human beings. We would mention especially the human case of Held and Goldbloom[3] and two of the cases which we previously reported in which the downward progress of the patient extended over a period of one to three weeks; and in which the patients themselves had failed to promptly recognize a serious condition.[1] This is to be contrasted with arterial occlusion proper in which the usual fulminating syndrome brings the patient rapidly to the doctor and in which the condition is recognized promptly as being surgical.

Dog 4 in Table III, which had recovered completely and which was killed on the thirty-seventh day, was quite interesting. The 10-inch loop which had been involved in venous occlusion showed a most distinct demarcation and discoloration. The gut which had been involved in occlusion presented a medium dark, distinct brown, somewhat streaked color over the entire wall surface, contrasting vividly with the color of the adjacent good gut. The wall itself had recovered fairly well, though grossly some fibrosis seemed present. We assumed that the discoloration was due to pigment from the broken-down blood cells of the hemorrhagic infarction and edema which followed the venous occlusion.

TABLE II

VENOUS OCCLUSION LOOPS (GUT LUMEN NOT OBSTRUCTED)
(12 TO 16 INCHES)

DOG NUMBER	SERIAL NUMBER	DATE OPERATED UPON	LENGTH OF LOOP INVOLVED	RESULTS	REMARKS
1	S10-D37	10/11/38	12 in.	Found dead 10/13/38	Gut reddish purple; no perforations or general peritonitis
2	S10-D36	10/11/38	12 in.	Found dead 10/13/38	Gut reddish purple; no perforations or general peritonitis
3	S10-D26	9/16/38	12 in.	Found dead 9/17/38	Not perforated; black
4	S10-D31	9/19/38	13 in.	Found dead 10/16/38	Was reopened 9/26/38; Grade 2 adhesions to area, of mesentery and omentum; collateral circulation being established; adhesions freed; found dead 10/16/38 apparently from mechanical obstruction of adhesions; gut loop blue
5	S10-D39	10/24/38	13 in.	Found dead 10/27/38	No peritonitis; gut dark, necrotic
	Microscopic Findings Hemorrhagic infarction, necrosis, or postmortem autolysis				
6	S10-D38	10/24/38	14 in.	Found dead 10/27/38	Fibrinous exudate about loop; gut black; some fluid in abdomen; no general peritonitis or ileus
	Microscopic Findings Hemorrhagic infarction, complete necrosis of mucosa; little inflammatory change; marked venous congestion; large saprophytic rods in otherwise normal mesenteric arterioles				
7	S10-D34	10/10/38	15 in.	Found dead 10/12/38	Loop reddish purple; no perforations
8	S10-D27	9/18/38	15 in.	Found dead 9/18/38	Free bloody fluid and gut perforated
9	S10-D29	9/18/38	16 in.	Found dead 10/11/38	Reopened 9/26/38; Grade 3½ adhesions omentum and mesentery; collateral circulation being established apparently; loop heavier, moderately deep dark red; all adhesions freed; died 10/11/38, cause unknown; involved loop definitely more blue than good gut; no perforations or peritonitis
10	S10-D35	10/10/38	16 in.	Found dead 10/26/38	On 10/24/38 dog was reopened under anesthesia. Some adhesions separated from the blue gut and animal closed; at autopsy two days later collateral circulation of omentum was being established
	Microscopic Findings Venous thrombosis with beginning organization; dilated lymph channels; desquamation arterial endothelium probably secondary; dubious thrombosis in few arterioles; no necrosis				
11	S10-D24	8/11/38	16 in.	Found dead 8/12/38	Gut deep reddish purple; distended

Died within 72 hr.	8	(72.7%)
Died in 16 days	1	(9.09%)
Recovered*	2	(18.1%)
Mortality		81.8%

*Because of the good general condition of Dogs 4 and 9 at the time reoperation was performed, the length of time which had elapsed before reoperation, the condition of the bowel as determined by reoperation, the fact that the animals died only after reoperation and severing of all adhesions and sources of collateral circulation which had been formed, we feel fully justified in classifying these two animals as recoveries.

TABLE III

VENOUS OCCLUSION LOOPS (GUT LUMEN NOT OBSTRUCTED)
(10 INCHES)

DOG NUMBER	SERIAL NUMBER	DATE OPERATED UPON	LENGTH OF LOOP INVOLVED	RESULTS	REMARKS
1	S10-D32	9/20/38	10 in.	Found dead 9/21/38	Gut black though not markedly liquefied; no peritonitis
2	S10-D30	9/19/38	10 in.	Found dead 9/21/38	General peritonitis, gut perforated
3	S10-D46	11/ 1/38	10 in.	Found dead 11/3/38	Loop gangrenous, perforated
4	S10-D47	11/ 2/38	10 in.	Killed 12/9/38	A sharp demarcation and brown color of entire loop; some collateral circulation through omental attachments
5	S10-D44	10/30/38	10 in.	Found dead 11/2/38	General peritonitis, gut gangrenous and perforated high jejunum
6	S10-D43	10/30/38	10 in.	Killed 12/9/38	Recovery with Grade 2 adhesions
7	S10-D40	10/29/38	10 in.	Killed 12/9/38	Grade 3 adhesions; gut wall somewhat sclerotic but recovered
8	S10-D45	11/ 1/38	10 in.	Found dead 11/10/38	Segment blue but not perforated or gangrenous; no peritonitis; cause of death uncertain
	Microscopic Findings Autolysis superficial layers mucosa; no other changes of note				
9	S10-D48	11/ 2/38	10 in.	Found dead 11/14/38	A few adhesions; distended and somewhat atonic; mucous membrane velvety with some exudate; no peritonitis; no ileus
	Microscopic Findings Superficial desquamation mucosa; no other changes of note				
10	S10-D42	10/30/38	10 in.	Found dead 10/31/38	Dark velvet loop, some exudate over thick mucous membrane; some red thin fluid in cavity; no distention or peritonitis
	Microscopic Findings Necrosis bowel wall; some hemorrhagic infarction and inflammatory change but not marked				
11	S10-D41	10/29/38	10 in.	Found dead 11/8/38	Loop adhesions; no peritonitis; cause of death uncertain; gut blue but not gangrenous or apparently greatly damaged
	Microscopic Findings Bowel wall thin; post-mortem (?) autolysis of mucosa				

Died within 72 hr.	5	(45.4%)
Died 10 to 12 days	3	(27.2%)
Recovered	3	(27.2%)
Mortality		72.7%

The specimen beautifully illustrated that a sharply demarcated and definite pathologic process had occurred over 10 full inches of gut and that yet the arterial supply of the gut had been able to function to the extent that the gut had remained viable until sufficient collateral circulation became established through the gut wall, omentum, and unobstructed lymphatics to allow recovery to occur.

In nearly all animals which recovered, the omentum actually did

establish a certain amount of collateral circulation. New vessels from it could be traced directly into the gut wall itself in some instances. In two or three animals which recovered, no evidence of collateral circulation outside the gut wall itself was present; and in these animals it appeared that with the 10 inches involved collateral circulation through the tissues of the gut wall alone seemed to compensate satisfactorily.

Some microscopic studies were done and are recorded in the tables. Necrosis and desquamation of the mucosa, and hemorrhagic infarction or degeneration of the gut wall tissues occurred in those animals which died promptly. The microscopic findings of Dog 10 given in Table II illustrate, we believe, the process which occurs in those cases in which damage has been rather severe but in which recovery takes place: "Venous thrombosis with beginning organization; dilated lymph channels; desquamation arterial endothelium probably secondary; dubious thrombosis in few arterioles; no necrosis."

SUMMARY

1. Forty-seven dogs were used in an experimental attempt to establish more definite clinical concepts of venous mesenteric occlusion by procuring a more precise correlation of the pathology of venous occlusion with clinical manifestations, prognostic and therapeutic indications.

2. Clinical cases do occur occasionally in which complete venous occlusions involving 20 inches or more of small gut run a relatively slow and deceptive course as compared to arterial occlusions; and recovery sometimes may occur without surgical intervention when such lengths are involved. The lymphatics allow for the egress of some edema; tissue pressure is relieved by seepage of fluid into the intestinal canal; collateral circulation tends to become established through the omentum and other structures which adhere to the involved site. Retrograde thrombosis into the arterioles does not necessarily occur; and the function of these latter may be surprisingly persistent, even when the gut wall is severely involved in the hemorrhagic infarction of venous occlusion.

Signs and symptoms of the potentially deceptive type of syndrome in venous mesenteric occlusions are discussed briefly and references to clinical case reports are given.

3. In these experiments no therapy, surgical or otherwise, was used. The animals were given water by mouth and a regular diet. In segments of small gut (dogs), 20 to 40 inches in length in which complete pure venous occlusions were produced suddenly (gut lumen not occluded), the mortality was 93.7 per cent. One animal out of sixteen spontaneously recovered (6.2 per cent). Two animals lived twelve days, illustrating the occasional slowness of the venous occlusion syndrome as compared to the invariably rapid exodus which follows prompt arterial occlusions of the same length.

In segments 12 to 16 inches in length the mortality was 81.8 per cent; recovery, 18.1 per cent; and one animal lived sixteen days before dying.

In 10-inch segments the mortality was 72.7 per cent; recovery, 27.2 per cent; and 27.2 per cent lived from ten to twelve days; 45.4 per cent died within seventy-two hours.

4. The small gut will recover spontaneously from larger areas of hemorrhagic infarction and pure venous occlusion than is commonly appreciated. Arbitrarily speaking, however, in venous occlusions involving completely 6 inches or more of the small gut wall, resection should be done if conditions permit.

We wish to express our appreciation to Dr. Albert DeGroat for the microscopic studies made. We also kindly thank Mr. George Cowan for the courtesies he has extended in assisting our photographic department in this and many other pieces of work.

REFERENCES

1. Babcock, W. W.: A Textbook of Surgery, Philadelphia, 1928, W. B. Saunders Co., p. 96.
2. Donaldson, J. K., and Stout, B. F.: Mesenteric Thrombosis, Am. J. Surg. **29:** 2, 208, 1935.
3. Douglas, John: Mesenteric Vascular Occlusion, Ann. Surg. **102:** 4, 636, 1935.
4. Held, I. W., and Goldbloom, A. A.: Rare Intraabdominal Cases, S. Clin. North America, p. 393, 1934.
5. Larson, Laurence M.: Mesenteric Vascular Occlusion, Surg., Gynec. & Obst. **53:** 54, 1931.
6. Meyer, Joseph L.: Mesenteric Vascular Occlusion, Ann. Surg. **94:** 88, 1931.

LOCAL LESIONS PRODUCED IN MICE BY STAPHYLOCOCCUS TOXIN AND BY TOXIN- AND NONTOXIN-PRODUCING STRAINS

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STAPHYLOCOCCUS toxin, when injected intradermally in rabbits, produces necrosis in the skin.[1, 2] Parker[3] studied this lesion microscopically; however, as she states: "No attempt has been made to carry out a complete or detailed study. In the section of a lesion taken one-half hour after injection no pathological alteration is discernible except slight edema which extends through the whole corium, and a scattering infiltration with a very few polymorphonuclear leucocytes and large mononuclear cells. Section of a lesion 24 hours old shows marked evidence of inflammation and beginning necrosis."

Stookey and Scarpellino,[4] in discussing staphylococcus infection of the skin, express the opinion that "the rapidly necrotic properties of the ulcers must be due to a powerful lytic agent. This we believe to be the exotoxin of the staphylococcus which, in our experience, is one of the most rapidly necrosing toxins elaborated by pathogenic bacteria."

It is well known that polymorphonuclear leucocytes localize about groups of staphylococci, either living or dead. This reaction is thought to be the result of positive chemotaxis. Different strains of staphylococci vary in their chemotactic action. Different species of animals also vary in their reaction to staphylococci.[5]

A lesion recently observed in the muscles of rabbits following the intravenous injection of a toxin-producing strain of staphylococci was different from those usually described as occurring locally with staphylococci. It was characterized by many necrotic muscle fibers surrounded by leucocytes. The histologic changes in the muscle were similar to those produced by staphylococcus toxin. This observation suggested that a study of the lesions produced by toxin- and nontoxin-producing strains of staphylococci might be of value in the interpretation of the pathologic lesions produced by staphylococci in the human being. The lesions produced by staphylococcus toxin in the subcutaneous tissue of mice were included in this study to compare the lesions produced by it with those produced by the organism.

MATERIALS AND METHODS

The staphylococcus toxin* used in this study contained 8,000 dermonecrotic units per cubic centimeter at the time of its preparation. It

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*The toxin was supplied through the courtesy of Lederle Laboratories.

was diluted 1:100 with physiologic saline solution immediately preceding the inoculation. Injections of 0.05 c.c. of this diluted toxin were made subcutaneously into each side of the abdomen of twenty white mice.

The toxin-producing strain was an aureus variant from a culture obtained in May, 1933, from the pharynx of a patient with agranulocytic angina. The lethal effect of this variant on rabbits was recently reported.[6] The ability to produce toxin was determined by the method of Biggers.[7] The supernatant fluid, after centrifugalizing the bacterial suspension obtained from washing the 48-hour growth from an agar slant 15 by 2.5 cm., contained enough hemotoxin in 0.05 c.c. to completely lyse 2 c.c. of a 1 per cent suspension of rabbit's cells. The nontoxin-producing strain of *Staphylococcus albus* was isolated in May, 1937, from a furuncle in the nose. No lysis occurred when 0.8 c.c. of the supernatant fluid from this culture was added to 2 c.c. of a 1 per cent suspension of red cells.

The staphylococcus strains were grown on agar slants for 18 to 24 hours immediately preceding inoculation. The bacteria were washed and suspended in a physiologic solution of sodium chloride. The suspensions of the organisms were grossly turbid and brought to approximately the same concentration. Injections of 0.05 c.c. of these suspensions were made subcutaneously on each side of the abdomen of white mice. Twenty were given the suspension of the nontoxin-producing strain and twenty-six the suspension of the toxin-producing strain. A No. 27 gauge needle was used. Two mice from each of the three groups were killed after 30 minutes, 1, 1.5, 2, 3, 4, 5, 6, 8, and 10 hours. Two mice inoculated with the toxin-producing strain of organisms were killed after 11 and 12 hours. The abdominal wall in the area of the inoculation was immediately removed from all the mice and placed in a 5 per cent solution of Zenker-acetic acid. The sections were prepared and stained with hematoxylin and eosin. Select sections were stained according to Giemsa's method.

Tissue from four uninoculated mice was used for the control.

EXPERIMENTAL

Effect of Staphylococcus Toxin.—The mice inoculated with the staphylococcus toxin appeared slightly less active than the controls. All survived the effect of the inoculum and were killed. The abdominal wall in the area of the injections was macroscopically edematous and hyperemic after 3 to 4 hours. The blood vessels were dilated on the peritoneal surface. Microscopically the vessels were dilated after 30 minutes. The subcutaneous tissues were edematous at this time. The muscle in the abdominal wall showed focal hyaline and nonstriated areas and necrosis after 90 minutes (Fig. 1*C*). The injury to the tissue apparently did not progress after the first 2 hours. Polymorphonuclear leucocytes were the characteristic cells present. After 30 minutes these cells lined the

lumen of the dilated vessels and were present in the adjacent tissue. Only a few leucocytes were found in the edematous and necrotic tissue during the first three hours after inoculation (Fig. 2*A*). Many of these

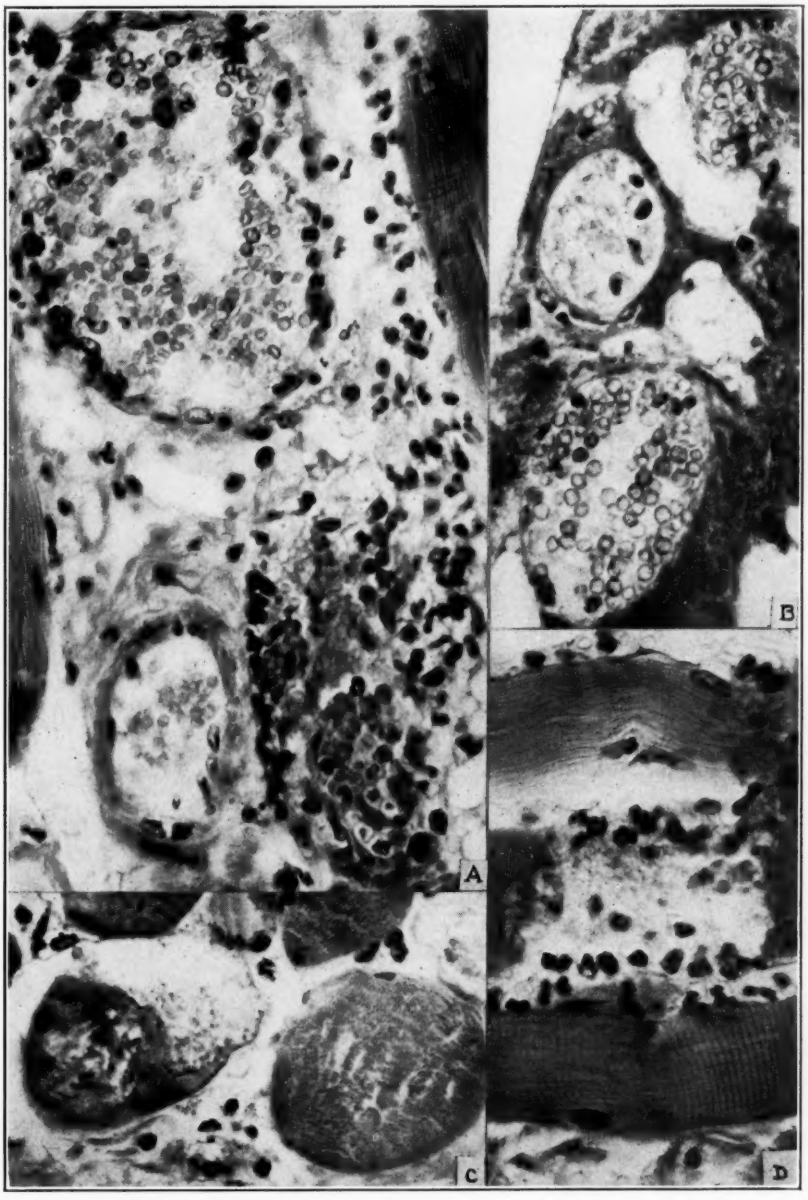

Fig. 1.—*A*, Polymorphonuclear leucocytes are present along the wall of these vessels and in the adjacent tissue. A toxin-producing strain of staphylococci was inoculated subcutaneously 30 minutes previously. This section is separated by normal tissue from the bacteria which are not shown here. *B*, A portion of the same tissue as shown in *A*. A solid mass of staphylococci surrounds the blood vessels. There are no leucocytes present. Apparently leucocytes are lysed very quickly by the toxin. *C*, Necrosis of the muscle in the abdominal wall 1½ hours following the injection of 0.05 c.c. of a 1/100 dilution of staphylococcus toxin. Note the absence of a leucocytic reaction. *D*, Necrosis of muscle in abdominal wall six hours after injecting the same amount of toxin as in *C*. Leucocytes are present about the debris. (Hematoxylin-eosin, ×600.)

cells during this time were pyknotic and lysed. After 6 hours the leucocytes appeared more numerous and better preserved. At this time they seemed to be in greater numbers about the necrotic muscle tissue (Fig. 1*D*).

Effect of the Nontoxin-Producing Strain of Staphylococci.—The mice appeared normal following the inoculation. The early lesions were

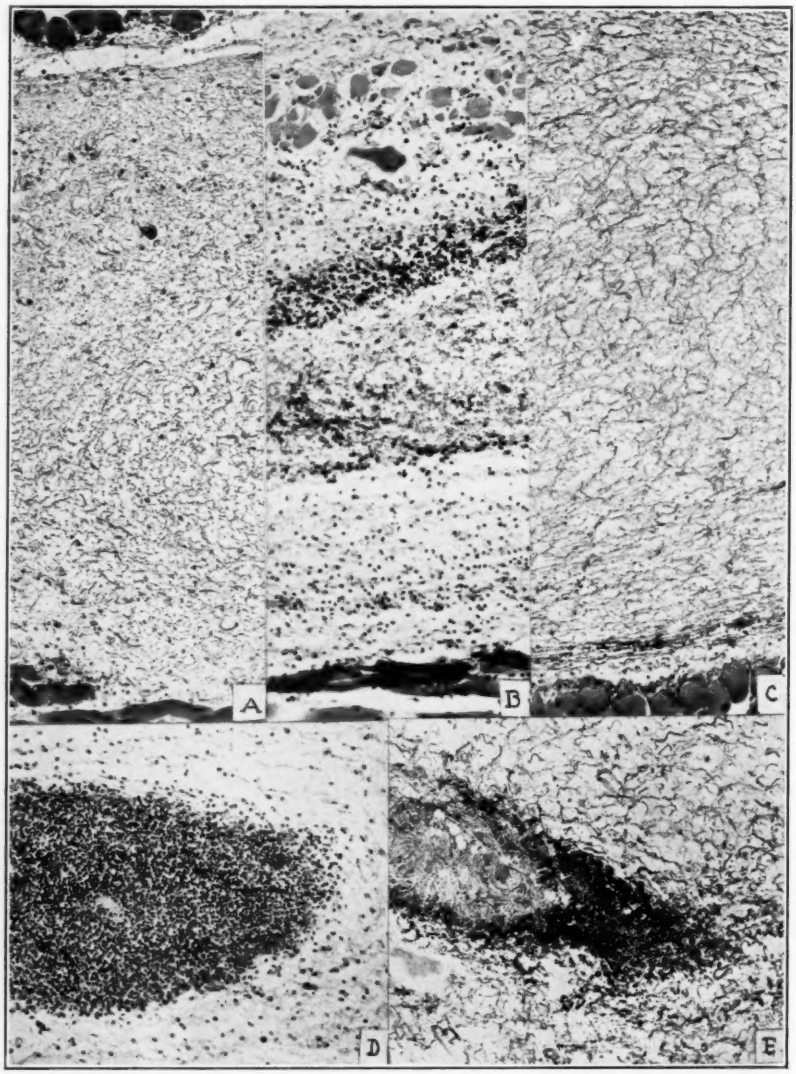

Fig. 2.—*A*, The tissue is edematous and few leucocytes are present. Many of the latter are degenerated. A 0.05 c.c. volume of a 1/100 dilution of staphylococcus toxin was injected 3 hours previously. *B*, The tissue is edematous. The area is infiltrated with leucocytes. The latter are surrounding and phagocytizing the bacteria. A 0.05 c.c. volume of the nontoxin-producing strain of staphylococci was injected 3 hours previously. *C*, The tissue is edematous. Only a few leucocytes are present and a majority of these appear to be injured. A toxin-producing strain of staphylococci equal to that given in *B* was injected 3 hours previously. *D*, The leucocytes have localized in the area previously occupied by the staphylococci and they have almost completely phagocytized the latter. The same suspension of organisms was given here 8 hours previously as in *B*. *E*, There are no leucocytes about this group of toxin-producing staphylococci. The same suspension of bacteria was given here as in *B*, 8 hours previously. (Hematoxylin-eosin, ×100.)

similar to those which occurred with the toxin. The bacteria were more or less localized in the subcutaneous tissue. The muscle never showed any evidence of necrosis and the subcutaneous tissue showed only a small amount at the end of the experiment. The polymorphonuclear leucocyte was the characteristic cell in the inflammatory process. After 30 minutes these cells lined the lumen of the dilated vessels and infiltrated the adjacent tissue. As time progressed, the leucocytes appeared to migrate toward the bacteria. After 3 hours the leucocytes had collected about the organisms and infiltrated the adjacent tissue (Fig. 2*B*). They were phagocytizing the bacteria. After 8 hours the leucocytes had invaded the entire area previously occupied by the bacteria and practically all the latter were phagocytized (Fig. 2*D*). Little change occurred in the reaction during the last two hours of the experiment.

Effect of a Toxin-Producing Strain of Staphylococci.—A majority of the twenty-six mice that received these organisms were weak and would not eat after 3 hours. Four of the group died 5 to 10 hours after the injection. Pathologic studies were made only on the mice that were sacrificed.

Macroscopically, the abdominal wall in the areas injected was edematous and hyperemic after three hours. As the experiment continued, the edema progressively spread to involve the greater portion of the wall. After 6 to 8 hours the skin was pale yellow in color. Microscopically, the changes after 30 minutes were similar only in few respects to those in which the nontoxin strain of staphylococci were injected. Polymorphonuclear leucocytes were present in the tissues about the blood vessels in the areas where there were no bacteria (Fig. 1*A*). Only a few leucocytes ever infiltrated the zone occupied by the organisms and they apparently were destroyed very rapidly (Fig. 1*B*). Phagocytosis was not a conspicuous process at any time during this experiment.

Although it was impossible to completely control the site of inoculation, there seems to be no doubt that the toxin-producing bacteria invaded very quickly the surrounding tissues. The leucocytes failed to localize the organisms after 8 hours as they did with the nontoxin-producing strain of staphylococci (Fig. 2*E*). Extensive necrosis occurred after 12 hours. Leucocytes invaded the wall of some of the smaller blood vessels and apparently some of the latter were thrombosed. Injured and degenerated leucocytes infiltrated the tissue in the abdominal wall. Staphylococci and leucocytes were present along the peritoneal surface. The corium and the squamous epithelium were necrotic in some areas.

DISCUSSION

The local lesions which occur following the local injection of a toxin-producing strain of staphylococci are very different from those which follow the local injection of a nontoxin-producing strain. The former

are characterized by a diffuse spread of the bacteria and extensive necrosis; the latter show essentially no necrosis and complete localization of the bacteria after 8 hours.

The cellular defense of the body, in the presence of a toxin-producing strain of staphylococci, apparently can play only a secondary role. Polymorphonuclear leucocytes respond to this type of infection; however, only a few are present about the bacteria (Fig. 1*B*). The toxin produced by the bacteria very quickly destroys the leucocytes. The organisms apparently spread rapidly in the absence of a cellular reaction.

The presence of leucocytes about the necrotic tissue after 3 hours in those mice inoculated with only staphylococcus toxin suggests that either all the toxin unites with the tissue or that it is at least adsorbed on the tissues by this time and that the subsequent cellular reaction is the response to the debris.

Leucocytes are not a conspicuous feature about the necrotic tissue in the mice injected with the toxin-producing strain. Apparently this is the result of the destructive action of the toxin which is continually formed by the staphylococci.

Van de Velde in 1894 observed the absence of leucocytes and the multiplication of bacteria in the pleural exudate following the local injection of a virulent strain of staphylococci in the rabbit.[8] A similar process apparently occurs in the subcutaneous tissue of mice following the local inoculation of a toxin-producing strain of staphylococci. Necrosis of muscle 30 minutes after the injection of the toxin-producing strain of staphylococci shows the rapidity with which this bacterium can produce toxin. This necrotic tissue, in the absence of phagocytic leucocytes, apparently serves as an excellent medium for the growth, multiplication, and spread of staphylococci.

The demonstration of the local effect of staphylococcus toxin produced in vivo and the presence of lesions produced by toxin in other organs[9] suggest that specific antitoxin is indicated in the treatment of lesions produced by some strains of staphylococci. Recently it was shown that staphylococcus antitoxin given intravenously to rabbits prolonged the period of survival following the intravenous injection of the same toxin-producing strain of staphylococci as used in this experiment.[6]

SUMMARY

The lesions which follow the local inoculation of a toxin-producing strain of staphylococci are very different from those which follow the injection of a nontoxin-producing strain of staphylococci.

Staphylococcus toxin is formed very rapidly in vivo and apparently unites quickly with striated muscle and leucocytes. It necrotizes muscle and lyses white blood cells within 30 minutes.

The organisms from toxin-producing strains of staphylococci apparently grow, multiply, and spread very rapidly through the necrotic tissues.

Polymorphonuclear leucocytes are the characteristic cells present in the tissues when either staphylococcus toxin or a toxin- or a nontoxin-producing strain of staphylococci is inoculated into the subcutaneous tissue of mice. Leucocytes phagocytize the bacteria very quickly from nontoxin-producing strains of staphylococci. Only a small amount of phagocytosis occurs, however, with a toxin-producing strain.

REFERENCES

1. Nelis, M. P.: Contribution à l'étude de la toxine staphylococcique et de ses dérivés, Bull. Acad. roy. de méd. de Belgique, 539-568, 1935.
2. Von Linzelsheim, W.: Aetiologie und Therapie der Staphylokokkeninfectionen, Berlin and Vienna, 1900, Urban & Schwarzenberg.
3. Parker, Julia T.: The Production of an Exotoxin by Certain Strains of Staphylococcus Aureus, J. Exper. Med. **40**: 761-772, 1924.
4. Stookey, Paul F., and Scarpellino, Louis A.: Dermonecrotic Properties of Staphylococcus Filtrates, J. South. M. A. **28**: 148-152, 1935.
5. Kolle, W., and Hetsch, H.: Die Experimentelle Bakteriologie und die infektionskrankheiten, Berlin, 1922, Urban & Schwarzenberg.
6. Rigdon, R. H.: The Effect of Staphylococcus Antitoxin on Rabbits Given Broth Cultures of Staphylococci Intravenously, J. Lab. & Clin. Med. **23**: 159, 1937.
7. Biggers, J. W., Boland, C. R., and O'Meara, R. H. O.: A New Method of Preparing Staphylococcus Hemolysin, J. Path. & Bact. **30**: 271, 1927.
8. Rigdon, R. H., and Leff, W. D.: A Gastro-Intestinal Lesion Associated With Staphylococcic Infection in Man, Arch. Path. **21**: 298-307, 1936.
9. Rigdon, R. H.: Some of the Experimental Lesions Produced by Staphylococcus Toxin, Read Before Section on Pathology, Southern Medical Association, November, 1938.

THE IMPORTANCE OF EARLY STUDY FOR METASTASES IN TUMORS OF THE BLADDER

Illustrative Cases

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WE BELIEVE the incidence of clinically recognizable metastases from bladder tumors is distinctly higher than one would gather from a study of the literature. References to metastases from bladder tumors are to be found in isolated case reports, which tend to emphasize the rarity of the condition and give the impression that, when metastases do occur in carcinoma of the bladder, they occur very late or in the end stages of the disease. A few reports, based on autopsy findings, have been thought to overestimate the frequency of metastases from bladder tumors. It generally has been felt that carcinomatosis is found much less often than in cancer of the breast, stomach, and prostate.

Cunningham (1931) analyzed 411 cases of carcinoma of the bladder reported in the literature and found metastases in 32.3 per cent. Spooner (1934) analyzed autopsy records in 163 cases of bladder tumor. Metastases occurred in 49 (29 per cent), but the clinical diagnosis of metastasis was made in only 4 cases. In 24 cases the symptoms were of less than two years' duration, and 19 patients had symptoms for less than one year.

Colston and Leadbetter (1936) found in 98 cases of infiltrating tumors of the bladder, which came to autopsy, 61.1 per cent which showed metastases. Many of these represented the end stage of cancerous disease of the bladder, but a considerable number did not, since many patients had been operated upon for extensive removal of the tumor. After retroperitoneal lymph gland involvement, the most frequently involved organs were the liver, lungs, and bone. Bone involvement was of frequent occurrence; namely, 18.1 per cent in 57 cases. This is noteworthy because from reports in the literature metastasis to bone is considered rather infrequent. The liver was involved in 41.8 per cent, and the lungs in 25.4 per cent. Involvement of the lymph glands was common, 72.7 per cent. The remainder of the metastases were distributed through a variety of organs.

In view of the high incidence of metastases to the lungs and bones, it is clear that x-rays of these organs should be a routine procedure in cases

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of bladder tumor. Careful palpation of the abdomen should be made to outline any enlargement or irregularity of the liver, and in selected cases exploratory laparotomy should be considered in an attempt to ascertain the status of liver enlargement as well as to palpate for enlarged glands in the retroperitoneal and iliac regions. Intravenous urography is of value in determining, by filling defects, the location and size of tumors within the bladder, and particularly in demonstrating lesions of the upper urinary tract resulting from tumors of the bladder.

Such careful studies for metastatic lesions become increasingly important as more radical surgery for cancer of the bladder is attempted. Although there is widespread use of high voltage x-ray therapy in the treatment of both early and late cases of carcinoma of the bladder, regardless of the grade of malignancy, opinion is divided in regard to the efficacy of such treatment. We believe that radical surgery still offers the best hope of ultimate cure in suitable cases. In infiltrating tumors involving the base and vesical orifice, radical surgery, involving transplantation of the ureters to either the skin or bowel, with cystectomy, has become increasingly important. Moreover, in certain cases with tumors situated around the trigone and vesical orifice and invading the prostate gland, a radical perineal operation, such as an extension of Young's radical operation for cancer of the prostate gland, can be performed. Before such radical surgery is attempted, it is absolutely necessary to do all in one's power to eliminate the possibility of either local or distant metastases.

Transurethral methods of treatment of bladder tumors have led to a general negligence on the part of urologists in searching carefully for metastases and have given rise to the opinion that metastasis from bladder tumors is rare and usually occurs only late in the course of the disease. Further support for this attitude is gained from the fact that many so-called benign papillomas of the bladder, which are potentially malignant, are not regarded with sufficient suspicion.

It is our purpose to report here 13 cases of papillary carcinoma of the bladder in order to show that clinically detectable metastases from bladder tumors are of rather frequent occurrence. These cases are not the only ones that developed metastases or had metastases at the time that they were under treatment, but these cases are the ones which were proved either clinically or at autopsy.

It is interesting to note that most of these cases were papillary types of bladder tumors, all of which probably were infiltrating, but some of which were small and showed little evidence of infiltration, even at the time of death. Metastasis occurred in some instances when the local tumor was small and apparently amenable to conservative therapy in the way of transurethral methods, or certainly radical surgery, and in some cases the primary tumor was thought to have been controlled or even eradicated. In several instances metastasis occurred relatively early

after the onset of the first symptoms. In one case bilateral transplantation of the ureters and subsequent cystectomy were considered the procedures of choice until an x-ray of the chest showed a small, circumscribed nodule of metastasis in the left lung. The following case reports and illustrations show how widespread some of the metastatic lesions may be and emphasize the importance of a careful search for metastases in order to avoid radical operations which are doomed to failure because of metastases. In such cases transurethral methods, plus high voltage therapy, constitute the procedure of choice.

CASE 1.—B.U.I. No. 19447. C. C., a 76-year-old man, was seen March 19, 1930, complaining of nocturia, burning on urination, and hematuria, all symptoms of a gradual progressive type over a period of five years. Five years before, cystoscopy disclosed a papilloma of the bladder, which was fulgurated at another clinic. During the next three years, several fulgurations were done to control intermittent bleeding. Six weeks before admission the symptoms became much more severe, with marked urgency progressing to almost incontinence. The general physical examination was essentially negative. Cystoscopy revealed multiple papillomas of the bladder, described as being short, sessile, papillary, nonulcerative tumors, and thought to be benign. Fulguration resulted in considerable alleviation of the symptoms. The patient returned nine months later with the same complaints. He had lost much weight and the hemoglobin was only 20 per cent. Cystoscopy showed a large papillomatous tumor, which appeared to be infiltrating, occupying the anterior wall of the bladder. Active bleeding was controlled by fulguration. The patient was given a blood transfusion, but failed quickly and died of cardiac failure ten days later. Autopsy revealed a papillary carcinoma of the bladder with extension to the right ureter and renal pelvis, and metastases were present in the liver which was found to be studded with nodules.

CASE 2.—B.U.I. No. 19888. G. H., a 54-year-old white male, was seen Aug. 12, 1930, complaining of burning and pain on urination, nocturia, and hematuria of five months' duration. Physical examination was essentially negative. Cystoscopy revealed a flat, infiltrating tumor, estimated to be 3.5 by 4 cm., covered with short papillary projections, involving the left side of the trigone, and adjacent left lateral wall of the bladder. Treatment consisted of surface application of radium and deep x-ray therapy. He was discharged one month later, but was readmitted in three months, complaining of pain in the suprapubic and perineal regions radiating down the posterior aspect of the left thigh and leg. On cystoscopy no definite tumor was visible. An x-ray of the pelvis showed a metastatic tumor involving the body of the fourth lumbar vertebra and the top of the sacrum. Deep x-ray was given as a palliative measure, with improvement of the pain, but subsequent x-ray revealed a large metastatic area in the left ischium (Fig. 1) as well. The patient died six months after his first admission. No autopsy was obtained.

In this case the symptoms had been present only five months before the patient came to the hospital. No biopsy was obtained, but the appearance of the tumor was such as to necessitate the diagnosis of papillary carcinoma.

CASE 3.—B.U.I. No. 20157. W. G., a 67-year-old white male, was admitted first on Nov. 12, 1930, complaining of frequency, nocturia, hematuria, and pain in the back for six months. The general physical examination was negative. Cystoscopy showed on the right half of the trigone a solid, sessile, lobulated tumor, 3 to 4 cm.

in diameter, with evidence of infiltration about its base. Suprapubic resection of the intravesical mass was carried out with the high frequency current and radium needles were implanted in its base and about its periphery. Microscopic sections of the tumor revealed an infiltrating papillary carcinoma. He was greatly improved at time of discharge, seven weeks after operation. He was readmitted three months later complaining of a suprapubic urinary fistula, frequency and burning on urination, and hematuria. Plain x-ray of the pelvis showed undoubted metastasis in the right ischium and pubis, which had been clear prior to operation. The patient was improved on discharge. No further records on the case were available.

Fig. 1.—Flat plate of pelvis (Case 2), showing large area of osteolytic metastasis in the left ischium.

Case 4.—B.U.I. No. 20998. C. C., a 52-year-old white man, admitted Sept. 24, 1931, complained of frequency and urgency of urination and hematuria, which had been present intermittently for two years. General physical examination revealed evidence of marked weight loss; dullness and impaired breath sounds in the left posterior lung field; and a palpable mass, about the size of a lemon, in the left side of the abdomen opposite the umbilicus. Renal function tests were normal. X-ray of the chest showed numerous circumscribed areas scattered throughout both lungs, characteristic of metastases. Gastrointestinal series was negative, except for displacement of the stomach due to what was thought to be an extragastric mass. Cystoscopy showed an infiltrating papillomatous tumor, with a broad base, about the size of a half dollar, located on the anterior wall. Treatment consisted simply of fulguration and the surface application of radium to destroy the local growth and relieve the symptoms. The patient died at home; no autopsy was obtained.

This case is an excellent example of widespread metastases from a small tumor of the bladder which, except for the presence of the metastases, certainly would have been considered amenable to either transurethral methods or suprapubic excision. If the metastatic lesions in the chest and the abdominal mass had been smaller, the metastases might easily have been missed.

CASE 5.—B.U.I. No. 21474. M. F., a 54-year-old man, admitted on April 6, 1932, complained of painless hematuria which had been present intermittently for a period of three years. He had been treated elsewhere, one year before, for a papilloma of the bladder. He was admitted in coma. Because of a distended bladder, he was catheterized and a decompression apparatus set up. General physical examination revealed a moderate cardiac enlargement with systolic murmurs at the apex and base. The blood pressure was 190/90. The blood urea was 56 mg. Cystoscopy could not be carried out, and the patient died a few hours after admission. Autopsy revealed a small papillary carcinoma of the bladder, 1 cm. in diameter, with extension to the region of the seminal vesicles and multiple metastases in the liver. There was a benign prostatic hypertrophy, but no renal disease was present.

This is another example of a very small infiltrating papillary carcinoma of the bladder with extensive metastases.

Fig. 2.—Flat plate of pelvis (Case 6), showing the marked bone destruction due to metastasis from a carcinoma of the bladder.

CASE 6.—B.U.I. No. 21499. M. E., a 46-year-old man, admitted May 2, 1932, complained of a bladder tumor which had been known to be present for one and one-half years and which had been treated by fulguration. The symptoms on admission were frequent and painful urination. The general physical examination, except for a hypertension of 182/110, was essentially negative. The blood urea was 36 mg. Cystoscopy revealed a solid, lobulated tumor springing from the bladder wall in the region of the right ureteral orifice, involving the right half of the trigone and extending up on the right lateral wall to the region of the fundus of the bladder. At suprapubic operation the intravesical tumor mass was fulgurated and coagulated thoroughly and radium needles implanted in its base, a total of 2,900 mg. hr. of radium being given. Microscopic examination of the material removed at operation showed papillary carcinoma. The postoperative course was stormy, but he was discharged in six weeks, greatly improved. During the next

three years he was readmitted several times for the purpose of removing vesical calculi which kept forming on the surface of the radium burn. Three years subsequent to his first admission, the patient noted pain and stiffness in the right hip and thigh. X-ray revealed evidence of osteolytic metastasis to the acetabulum, ischium, and superior ramus of the pubis. Careful study of the bladder showed no evidence of recurrence of the tumor growth. Deep x-ray therapy was given to the region of the right hip with alleviation of the pain. The patient was readmitted one year later because of excruciating pain in the right hip, thigh, and knee. Examination at this time showed marked enlargement and deformity of the right side of the pelvis and upper thigh. X-ray showed the right side of the pelvis to be almost completely destroyed by an osteolytic metastatic lesion (Fig. 2). Cystoscopy releaved an extensive recurrence of the bladder tumor involving the left half of the trigone and the bladder floor. The patient died one month later; no autopsy was obtained.

CASE 7.—B.U.I. No. 22575. R. W., a 62-year-old man, admitted Oct. 10, 1933, complained of frequent urination and intermittent hematuria over a period of two years. The general physical examination was essentially negative except for palpable enlargement of the liver. Rectal examination was negative. On cystoscopy there was seen a large, shaggy, papillary tumor involving the vesical orifice and extending over the left half of the trigone up to the left lateral wall, obscuring the left ureteral orifice. X-ray of the pelvis revealed an osteoplastic metastasis in the right ilium. Cystogram showed an extensive filling defect involving the left lateral wall of the bladder. Biopsy of the growth revealed papillary carcinoma. Fulguration of the growth was carried out on two occasions with considerable relief of symptoms. The patient was readmitted two months later, at which time considerable decrease in the size of the tumor was noted. The patient received a series of radium applications to the base of the tumor. Ten months after the first admission, x-ray revealed changes in the right ilium and pubis which were characteristic of metastasis. Cystoscopy at this time showed a recurrence of the bladder tumor and evidence of infiltration. The patient was not seen thereafter.

CASE 8.—B.U.I. 23362. A. S., a 34-year-old man, admitted to the orthopedic service on Oct. 11, 1934, complained of pain in the left hip of two months' duration, and intermittent hematuria over a period of one year. Examination revealed an emaciated man who looked chronically ill. There was localized tenderness in the region of the left sacroiliac joint, but no mass or swelling could be made out. Cystoscopy revealed a large, papillary, pedunculated tumor springing from the region of the left ureteral orifice. The tumor was nonulcerative and did not appear infiltrative. The renal function tests were normal. Intravenous urography revealed a hydronephrosis on the left side. X-ray of the bones of the spine and pelvis showed changes which were interpreted as being an extensive arthritis of the left sacroiliac joint rather than metastasis. Suprapubic cystostomy was performed and the tumor, which was found to be 7 cm. in diameter, was excised and the base fulgurated. The pathologic report was papillary carcinoma. Convalescence was uneventful, but pain in the left sacroiliac joint continued. X-ray of the pelvis was repeated and showed partial destruction of the left portion of the ilium due to metastasis. This was proved by biopsy of the ilium to be metastatic papillary carcinoma. The patient was readmitted two months later because of increased pain in the left hip. Cystoscopy at this time showed no evidence of recurrence of the bladder tumor, but x-rays of the pelvis showed marked destruction of the left side of the top of the sacrum and ilium (Fig. 3) with some new bone formation. Deep x-ray of this region was given for control of the pain, but the patient died three months later, presumably due to further metastatic lesions, although no autopsy was obtained.

This case was exceedingly interesting in that we were not sure that this was ever an infiltrating tumor of the bladder; it may have been simply a superficial papillary pedunculated tumor. It also illustrates the point that a distant and perhaps widespread metastasis may occur from a relatively small and easily treated tumor of the bladder.

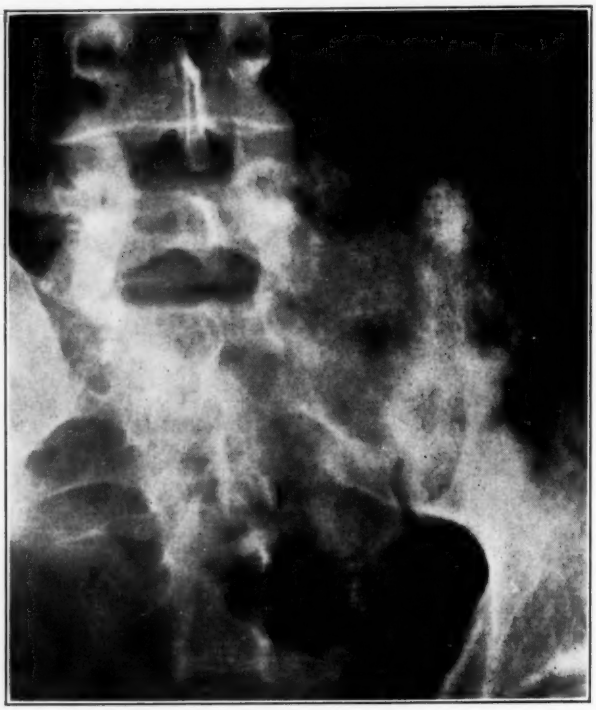

Fig. 3.—Flat plate of pelvis (Case 8), showing the osteolytic metastasis to the left sacroiliac joint from a papillary carcinoma of the bladder.

Case 9.—B.U.I. No. 24495. C. H., a 63-year-old white male, admitted Jan. 17, 1936, complained of hematuria for six months. The physical examination was negative. Cystoscopy revealed a nodular, infiltrating tumor on the left side of the bladder and around the vesical orifice. Intravenous urogram showed no function on the left side; the right kidney appeared to be normal. The cystogram showed a filling defect involving the left side of the bladder and the vesical orifice. The hematuria ceased after the cystoscopic application of radium, and the patient was discharged. He returned three months later, complaining of marked hematuria, weakness, and jaundice, at which time the liver was thought to be enlarged to five fingerbreadths below the costal margin, and was irregular and firm. A biopsy revealed papillary carcinoma. The blood urea was normal. The van den Bergh reaction showed a prompt biphasic response, 7.3 mg. per cent. The stools were clay colored and fatty. The patient was readmitted one month later in deep jaundice, with marked itching of the skin, weakness, and anorexia. Examination revealed an acutely ill, emaciated man, with the liver palpable at the level of the umbilicus, but with no ascites. Exploratory laparotomy disclosed a large, dark liver and a distended gall bladder and common duct. At the terminal portion of the common duct there was a large, nodular, fixed mass occupying the region of the head of the pancreas. The patient died shortly after operation. Autopsy revealed extensive carcinoma of the bladder

with metastases to the common bile duct, pancreatic ducts, and left adrenal, as well as left hydronephrosis and hydroureter. Microscopic examination of the metastatic sections revealed a papillary carcinoma quite similar to that in the bladder.

Although the local tumor in this case was extensive, nevertheless the case illustrates the necessity for considering metastases as the basis for untoward developments in a case, and this man died not from the local tumor but of metastatic lesions.

CASE 10.—B.U.I. No. 23833. C. S., a 47-year-old man, first seen on April 26, 1935, complained of frequency, nocturia, some dysuria and hematuria, which had been present for a period of eight months and which was gradually increasing in intensity. He also complained of pain radiating down the posterior aspect of the right thigh and leg. Physical examination was essentially negative. On rectal examination there was noted slight induration at the base of the prostate on the right side and also extending up into the posterior wall of the bladder. Intravenous urogram revealed a normal left kidney, but no dye appeared on the right side. Cystogram showed a large, irregular filling defect occupying practically the entire right half of the bladder. On cystoscopic examination the right side of the bladder was found to be occupied by an extensive, irregular, somewhat nodular, infiltrating tumor, which extended from the vesical orifice to the midline anteriorly and down over the right half of the trigone, obscuring the right ureteral orifice. Radium treatment, consisting of 600 mg. hr. of surface radium, was given, together with deep x-ray therapy. There was an immediate response. The pain in the right hip and leg, which had been extremely distressing, disappeared during the first week, and the urinary symptoms gradually disappeared during the course of the treatment. Rectal examination six weeks later showed that the induration at the base of the bladder had almost completely disappeared, and cystoscopic examination showed a remarkable change. The tumor had entirely disappeared except for a small, projecting mass of apparently viable tissue in the region of the right ureteral orifice, which was destroyed by means of the high frequency current. The patient returned three months later with symptoms and signs of increased intracranial pressure, thought to be due to a lesion on the left side of the brain. X-rays of the chest, pelvis, and skull revealed no evidence of metastases. Shortly thereafter a rather sudden exacerbation of all symptoms occurred and the patient became semicomatose. A circumscribed tumor was removed from the region of the third ventricle by Dr. Walter E. Dandy. The surgical pathologic report stated that it was obviously not a primary brain tumor and that it suggested very strongly a carcinoma arising from transitional epithelium. The patient did not survive. Autopsy showed no evidence of tumor anywhere except in the brain and the bladder. The right kidney was atrophic and hydronephrotic. The left kidney and ureter were normal. When the bladder was opened, there was no gross evidence of tumor, but there were two dense scars involving the right side of the trigone and the right lateral wall. Only two small areas of superficial ulceration were seen and it required serial sections through these areas to find any tumor.

This case was interesting: (1) in that it represented an unusual metastasis from any tumor; that is, tumor of the brain without any other evidence of metastasis elsewhere; and (2) because of the almost miraculous response to deep x-ray and radium of the large bladder tumor. Two other cases of the same type have been reported, one by Lower and Watkins and the other by McKay. The mechanism of such a metastatic deposit is a little hard to understand, but it must be that a few tumor cells

get into the blood stream and are carried through the pulmonary circulation to the brain. This case was reported in more detail by Leadbetter and Colston.

CASE 11.—B.U.I. No. 26450. G. H., a 49-year-old man, was admitted Dec. 9, 1937, complaining of a bladder tumor which had been diagnosed elsewhere in July, 1937, and treated by deep x-ray and fulguration without any results. The patient complained of great frequency, hematuria, anorexia, and loss of weight and strength. The general physical examination, except for marked malnutrition, was negative. On rectal examination the prostate was felt to be slightly enlarged, with induration at its base and in the region of the base of the seminal vesicles, which was thought

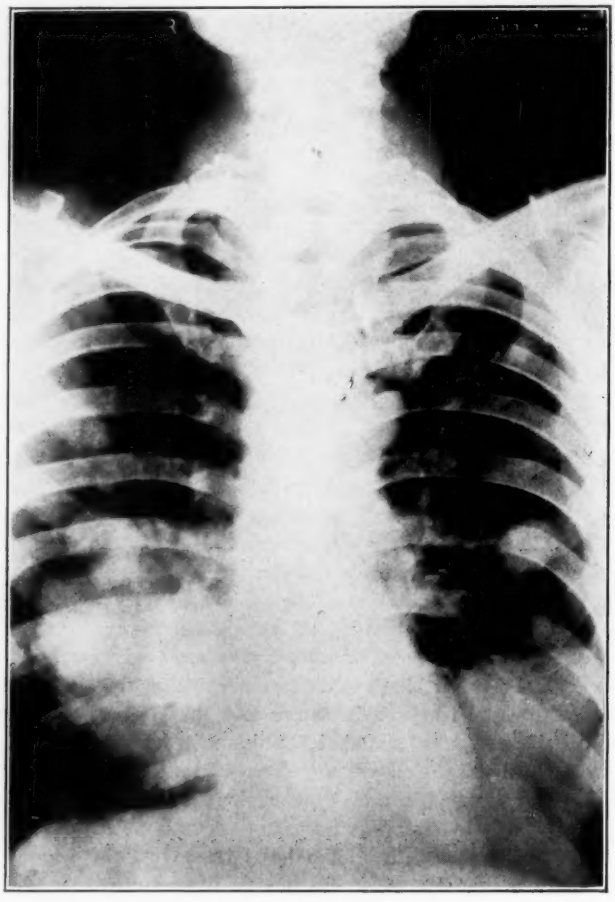

Fig. 4.—X-ray of the chest (Case 12), showing the widespread metastatic lesions in the lungs from the primary carcinoma of the bladder.

to indicate infiltrating tumor extending through the bladder wall. The laboratory studies were within normal limits. Intravenous urogram showed an early hydronephrosis on the right side; the left kidney appeared quite normal. Cystogram revealed an extensive filling defect involving the right side of the bladder. Cystoscopy showed a large, ulcerating tumor involving the right lateral wall of the bladder and the right half of the trigone. Transplantation of the ureters into the large bowel, followed by cystectomy, was considered, but x-ray of the chest showed a

clearly defined pulmonary metastasis. No biopsy of the tumor was obtained and the patient was discharged without treatment.

CASE 12.—B.U.I. No. 25360. T. W., a 50-year-old man, was seen in the outpatient department on Dec. 17, 1936, complaining of painless hematuria, usually initial in character, for a period of about one year. The general physical examination was negative. Blood studies and renal function tests were normal. Intravenous urogram showed both kidneys functioning well. Cystogram showed a filling defect on the right side of the bladder. Cystoscopy revealed a tumor in the region of the right ureteral orifice, described as a pedunculated papillary growth, 3 cm. in diameter, with evidence of fusion of the superficial papillae, slightly necrotic, and with a rather broad pedicle. The impression was noninfiltrating papillary carcinoma. Treatment consisted of fulguration and the application of 200 mg. hr. of radium to the surface of the tumor, following which the hematuria ceased and the patient was discharged, to be followed in the outpatient department. He was readmitted one month later, when the remaining tumor was again fulgurated and 200 mg. hr. of radium was applied. The patient was instructed to return in three weeks for further treatment, but he was not seen again until seven months later, at which

Fig. 5.—Flat plate of the pelvis (Case 12), showing the marked involvement of the bones of the right hip by the metastatic process from the carcinoma of the bladder.

time he stated that he had been passing bloody urine for several months. General physical examination and laboratory studies were negative. Cystoscopy showed, in the region of the right ureteral orifice, a large, sessile, superficially papillary, infiltrating tumor, about 3 cm. in diameter. Treatment again consisted of fulguration and the application of 200 mg. hr. of radium. One month later he had developed considerable pain in the right hip, but an x-ray of this region showed no evidence of metastasis. There was, however, a slight change in the hip joint which was interpreted as being due to arthritis. At this time the bladder tumor showed some evidence of extension and more infiltration, and 200 mg. hr. of radium was applied. Biopsy at this time showed papillary carcinoma. The patient was started on deep

x-ray therapy, which was continued in the outpatient department after his discharge. He was readmitted shortly thereafter complaining bitterly of pain in the right hip which radiated down the thigh toward the knee. Physical examination, aside from some obvious loss of weight, was negative, and there was nothing palpable about the hip or in the thigh which could account for his pain. Cystoscopy was again carried out and 200 mg. hr. of radium applied to the surface of the tumor. X-ray of the right hip showed early destruction about the hip joint characteristic of metastasis. Intravenous urogram showed hydronephrosis of the right kidney. His last admission was

Fig. 6.—Section from the bladder tumor (Case 12), showing a typical papillary carcinoma (low power).

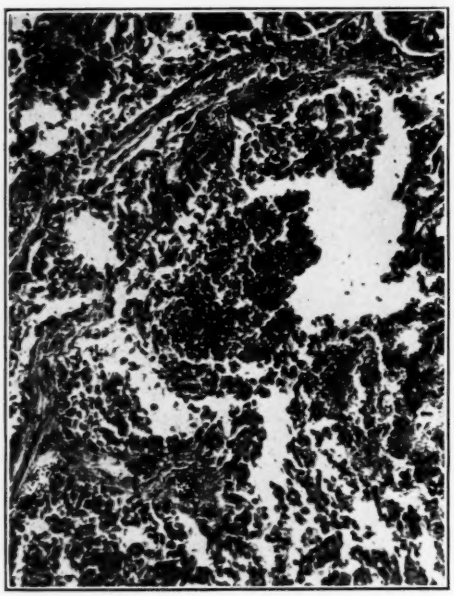

Fig. 7.—Section showing the tumor cells in the lung (Case 12) growing in a very wild and invasive fashion (low power).

two months later, at which time physical examination revealed marked emaciation, impaired percussion note, decreased tactile and vocal fremitus over the right chest below the sixth rib, and considerable atrophy of the muscles of the right thigh with great pain accompanying movement of the leg. X-ray of the chest showed numerous large metastases (Fig. 4) and that of the right hip showed increased destruction of the acetabulum (Fig. 5). The patient's course in the hospital was steadily downhill, but he lived a surprising length of time in view of the extensive metastases, death occurring three months after the last admission. Autopsy showed extensive

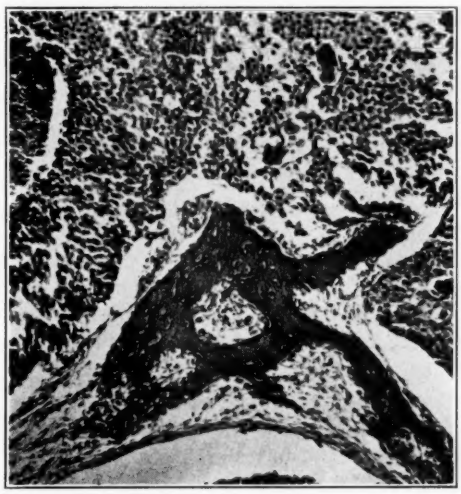

Fig. 8.—Section showing the metastatic tumor (Case 12) invading a vertebra and growing in its characteristic papillary formation (low power).

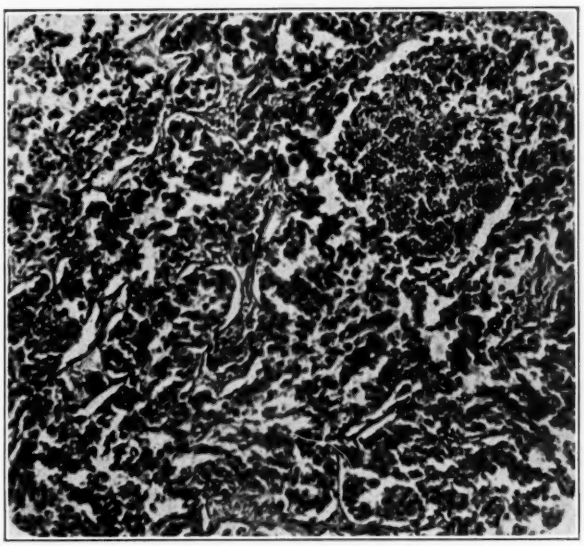

Fig. 9.—Metastatic tumor (Case 12) growing in the liver in characteristic invasive manner (low power).

metastases to the iliac glands, liver, both lungs, the mediastinal glands, the vertebrae, sternum, ribs, and pelvis. There was also a cystitis, a bilateral hydroureter, and hydronephrosis on the left side. On microscopic study all of the metastatic

lesions showed the same typical configuration of papillary carcinoma as seen in the original tumor (Figs. 6-9).

The widespread metastases from a relatively small, infiltrating, papillary carcinoma of the bladder make this case unusually interesting. It is also interesting to note that the metastases developed to a large extent after the onset of deep x-ray therapy. Whether or not this had anything to do with the incidence of metastasis would be hard to say, but it has been suggested by this and other cases.

CASE 13.—B.U.I. No. 26207. Mrs. C. E. K., a 65-year-old woman, was admitted Sept. 27, 1937, complaining of a bladder tumor which had been diagnosed elsewhere only a few days previously. Her symptoms had been present for several years, manifested by frequency, nocturia, and very slight hematuria. General physical examination was negative. Renal function tests were within normal limits. Cystoscopy showed a well-demarcated, globular, rather solid neoplasm, with superficial papillary processes, occupying the base of the bladder, involving the trigone in the midline, and extending backward and to the right, apparently involving the region of the right ureteral orifice. Biopsy showed a papillary infiltrating carcinoma. The tumor was fulgurated and surface radium applied on several occasions. Intravenous pyelogram showed a nonfunctioning right kidney and slight dilatation of the lower left ureter, but the left kidney appeared normal. Vaginal examination showed evidence of infiltration of the tumor into the vesicovaginal septum. Plain plate of the abdomen showed no evidence of metastasis to the bones of the spine or pelvis. She was symptomatically greatly improved on discharge. The patient was readmitted three months later complaining of chills and fever. The general physical examination revealed nothing of note. On cystoscopic examination the tumor appeared smaller but not greatly altered. In addition, several small, new tumors were noted, which were fulgurated. She was readmitted two months later complaining of pain in the right shoulder and left hip and intermittent attacks of fever and weakness. Physical examination showed no demonstrable changes in the bony framework, but x-rays of the right shoulder showed a rarefaction at the head of the right humerus characteristic of a metastatic lesion, and x-rays of the thoracic and lumbar spine revealed metastases. Cystoscopy showed a solid, globular tumor mass, involving the left half of the trigone and the left bladder base and extending down to the vesical orifice. Vaginal examination showed extensive infiltration into the pelvic tissues on the left side. The metastatic lesions were treated with deep x-ray with considerable relief of the pain. No treatment of the local tumor was carried out. The patient was discharged and the outcome is not known.

SUMMARY

The thirteen cases of bladder tumor with metastases, presented above in brief, were seen during a period of eight years at the Brady Urological Institute. We realize that they do not represent the total number of cases of bladder tumor in which metastases developed during this period, since many of the cases were not studied from this viewpoint and several of the others who died did not have post-mortem examination. It is of interest to note that all of our cases, with possibly one or two exceptions, were papillary carcinomas of the bladder.

We hope that this study will lead to a more general appreciation of the part played by metastases in cases of bladder tumors, and that in all

cases, before treatment is carried out, careful search by all means at our disposal will be made for a possible silent metastasis.

CONCLUSIONS

All bladder tumors should be regarded with high suspicion, because many apparently benign papillomas of the bladder eventually may recur as malignant, spreading tumors.

All cases should have an intravenous urogram to determine the function and gross condition of the kidneys.

Metastases may occur relatively early in the course of the disease and may be located at a considerable distance from the primary site without any evidence of lymphatic involvement, such as in the case of metastasis to the brain or to bone.

Metastases may occur in spite of the fact that treatment of the local tumor is progressing quite satisfactorily. This should indicate that a more frequent checkup of bladder tumor cases should be made so that we will not be lulled into a sense of false security while a possible metastasis is developing.

If radical surgery is to be considered, thorough x-ray study of the chest, the bones of the pelvis and spine, and the long bones, as indicated, should be obtained.

REFERENCES

Colston, J. A. C., and Leadbetter, W. F.: Infiltrating Carcinoma of the Bladder, J. Urol. **36**: 669-683, 1936.

Cunningham, J. H.: Tumors of the Bladder, J. Urol. **25**: 559-587, 1931.

Leadbetter, W. F., and Colston, J. A. C.: Brain Metastasis in Carcinoma of the Bladder, J. Urol. **38**: 267-277, 1937.

Lower, W. E., and Watkins, R. M.: Case of Primary Carcinoma of the Bladder With Metastasis to the Brain, Am. J. M. Sc. **167**: 434, 1924.

McKay, H. W.: Solitary Metastasis to the Brain From Carcinoma of the Bladder, Brit. J. Urol. **2**: 156, 1930.

Spooner, A. D.: Metastasis in Epithelioma of the Urinary Bladder, Tr. Am. A. Genito-Urin. Surgeons **27**: 81, 1934.

SCALENUS ANTICUS SYNDROME AND CERVICAL RIBS*

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SCALENUS anticus syndrome[1] is a symptom complex characterized by a brachial neuritis with or without vascular or vasomotor disturbance in the upper extremity. The pathologic condition is due to mechanical irritation or pressure[2] on the brachial plexus and subclavian artery produced by the anatomic relations of these structures to the scalenus anticus muscle and first thoracic rib (Fig. 1). This pressure was noted by Murphy[3] in 1915 while resecting a cervical rib, but he made no attempt to remove the pressure. Ten years later, Adson[4] performed and recommended scalenotomy instead of rib resection in cases of cervical rib. The good results of the new procedure, as reported by Adson, suggested to Naffziger[5] that the scalenus anticus muscle might be the cause of the symptoms when a cervical rib is suspected but cannot be demonstrated on roentgenograms. This group of cases with identical symptoms as seen in 5 to 10 per cent of cervical ribs[6] is recognized today as scalenus anticus or Naffziger syndrome.

The diagnosis of scalenus anticus syndrome is based upon the presence of a brachial neuritis, but there is no definite means of establishing the mechanical origin of the neuritis or the fact that the scalenus muscle is the cause of the compression. To confirm the diagnosis, Ochsner, Gage, and DeBakey[7] and Spurling and Bradford[8] have used oscillometric readings. We, however, have found the same characteristic readings in normal individuals. The frequent uncertainty in diagnosis, as well as the many conditions which may compress the brachial plexus, makes it unlikely that scalenotomy, the recognized treatment, will always give complete relief. Permanent changes may occur in the nerves, arteries, and extremity which will limit the relief to be obtained from operation. This is contrary to the unusually successful results reported by the above authors and others[8] who advise routine scalenotomy. Further, the fact that the symptoms are aggravated by faulty posture indicates that conservative treatment is frequently all that is necessary. In twenty cases of suspected scalenus anticus syndrome which we have examined, scalenotomy was performed on three patients. These three cases, which are reported herewith, were thoroughly examined by neurologists who confirmed the diagnosis and recommended operation.

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REPORT OF CASES

CASE 1.—Mr. J. S., aged 36 years, was referred because of an atrophied, cold, and deformed right hand (Fig. 2) which had been repeatedly diagnosed and treated as Raynaud's disease. Prior to 1931, he had been in excellent health and, except for the above condition, he has had no other complaints. There was no history of

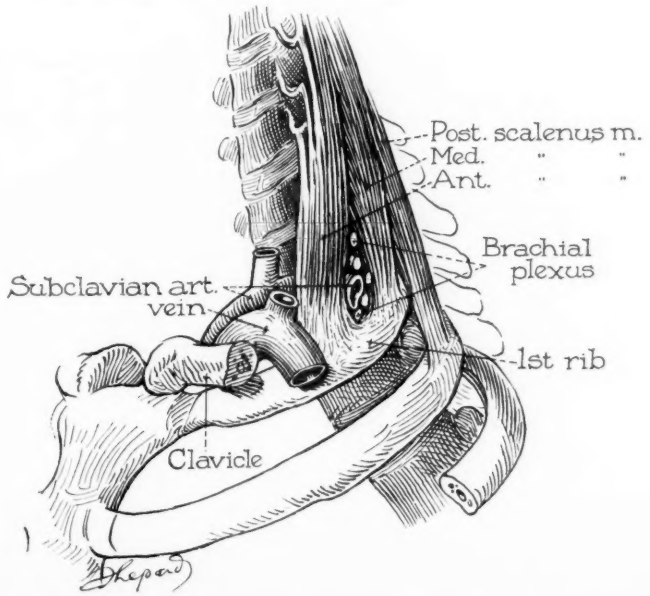


Fig. 1.—The anatomic relationship of the brachial plexus and subclavian artery to the scaleni and the first thoracic rib.

Fig. 2.—Deformed and atrophied right hand in case of bilateral cervical rib (Case 1). Resection of the right cervical rib failed to give symptomatic relief of the brachial neuritis. Eighteen months later scalenotomy was followed by marked relief of neurologic symptoms.

trauma. In 1931 he noticed several blue spots on the fingers of his right hand which were associated with moderately severe burning and sharp pains. Within two months the finger tips became ulcerated. The gangrenous finger tips completely healed within one week. This was followed by incomplete fixation of the interphalangeal joints, contraction deformities, and marked atrophy of the fingers. There was some improvement in the pain and coldness of the fingers, but for the past three years the condition has remained unchanged, still painful and markedly sensitive to cold.

Although the above symptoms were entirely unilateral, roentgenograms revealed large bilateral cervical ribs (Fig. 3). In spite of the fact that the left cervical rib was almost as large as the right, the left side was symptomless.

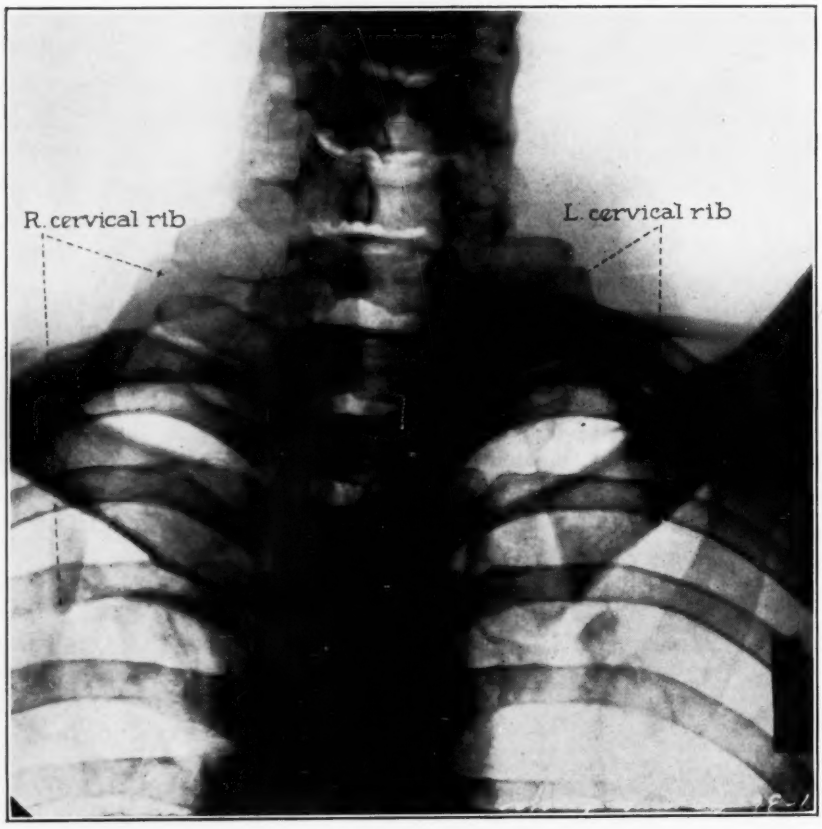


Fig. 3.—Roentgenogram of bilateral cervical ribs of case shown in Fig. 2 (Case 1).

Examination on March 24, 1936, revealed cold, atrophied, and deformed fingers of the right hand. The skin was cyanotic, atrophic, and shiny. The right radial, brachial, and subclavian artery pulsations could not be palpated. Definite tenderness was present in the right supraclavicular triangle. The average temperature of the fingers of the involved right hand was about 2° C. lower than that on the left hand. Complete neurologic examination of the extremity showed no sensory disturbances.

Oscillometric and peripheral temperature readings were made on both the right and left upper extremities in the sitting and reclining positions. The oscillometric readings on the right side were markedly reduced (less than 1) and varied but little

in changing from the sitting position to the reclining position. On the left, uninvolved side, the oscillometric index increased from 5.2 to 7.4 in the arm and from 2 to 3.4 at the wrist. In the reclining position the average temperature of the right (involved) fingers was 32.2° C., which was higher by almost 1° C. than on the left side. After sitting in a chair for one-half hour, the originally higher temperature on the right side dropped to 29° C., while on the left the average temperature declined from 31° C. to 30° C. Temporary novocain block of the ulnar nerve eliminated the above change in peripheral temperatures.

On April 20, 1936, resection of the right cervical rib was performed. No improvement in oscillometric readings or symptoms following the operative procedure.

On Oct. 25, 1937, the oscillometric readings, peripheral skin temperatures, and symptoms were unchanged from the initial examination made before rib resection. The same findings were obtained on Dec. 1, 1937. In the absence of symptomatic relief from the rib resection, scalenotomy was then decided upon.

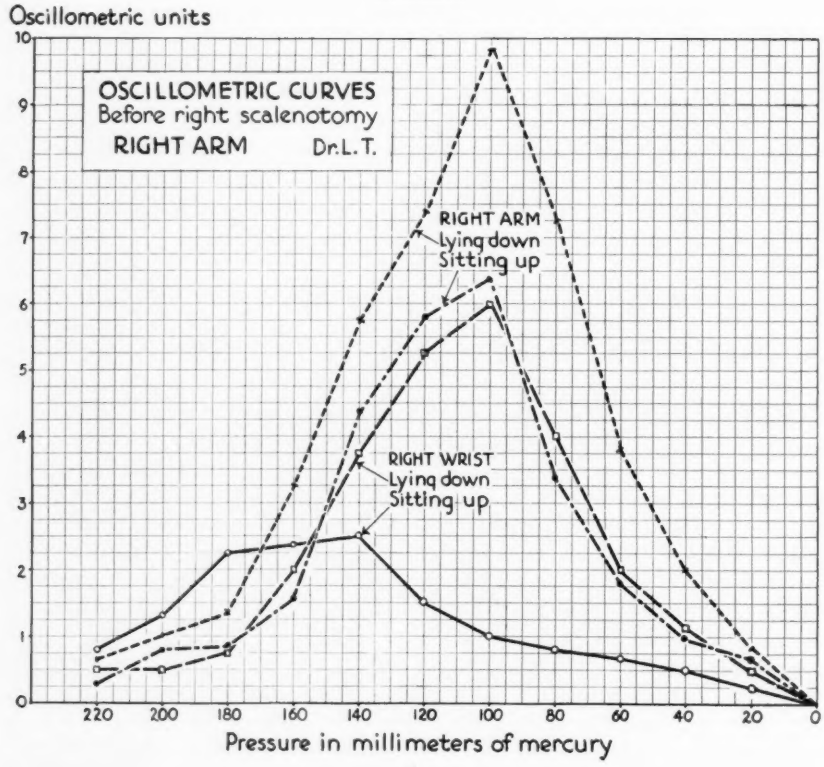
Right scalenotomy was performed on Dec. 16, 1937. The scalenus anticus muscle and the posterior fibrous sheath were markedly hypertrophied. Definite clinical improvement with relief of pain followed this operative procedure. Although the peripheral arterial pulsations did not return, there was a slight increase in the oscillometric index, and almost normal skin temperatures. Because of thrombosis in the subclavian artery, the normal peripheral pulsations were not restored.

Comment.—After more than five years of suffering with a unilateral condition of the right hand which was repeatedly diagnosed as Raynaud's disease, roentgenograms revealed extensive bilateral cervical ribs. Resection of the rib on the involved side gave no symptomatic relief. Oscillometric index did not vary with postural change, but this was due to thrombosis of the subclavian artery. More than eighteen months later, scalenotomy was followed by marked relief of the neurologic symptoms.

CASE 2.—Dr. L. T., aged 47 years, was examined because of weakness and numbness of the right fingers, hand, and forearm and mild atrophy of dorsal interossei muscles. Although similar symptoms were present to a slight extent on the left side, these were not troublesome compared to those on the right. No objective sensory disturbances were present. Moderate tenderness of the right scalenus anticus muscle was elicited by digital pressure, but there was no aggravation of the peripheral symptoms. In changing from the upright to the reclining position, a markedly increased oscillometric index of 3.5 was present on the right arm and only 1.5 on the left (Fig. 4). Cervical roentgenograms were negative. There was no history of trauma. Tenotomy of the right scalenus anticus muscle was performed on June 10, 1938.

Within forty-eight hours after the scalenotomy, there was definite improvement in the ulnar side of the hand, but the remainder of the hand rapidly became worse and was associated with atrophy and deformity of the small muscles of the hand. This became so severe that on Nov. 11, 1938, the right brachial plexus was explored through the original scalenotomy incision. No adhesions or abnormality were found.

Comment.—Although the diagnosis of scalenus anticus syndrome was confirmed by a number of neurologists, only temporary and partial relief followed scalenotomy. Subsequently, exploration of the supraclavicular triangle failed to reveal a cause for continuation and progress of the symptoms. The latest neurologic report indicates the presence of a cord lesion.



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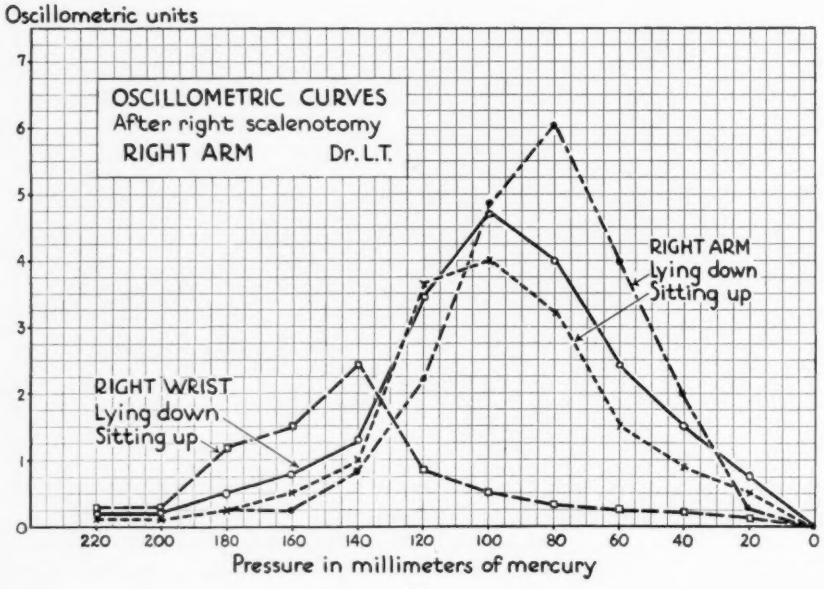
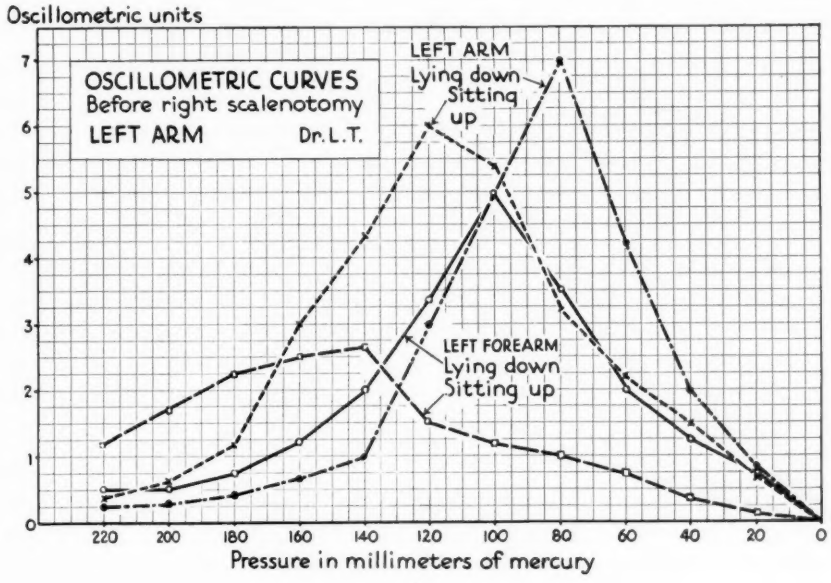


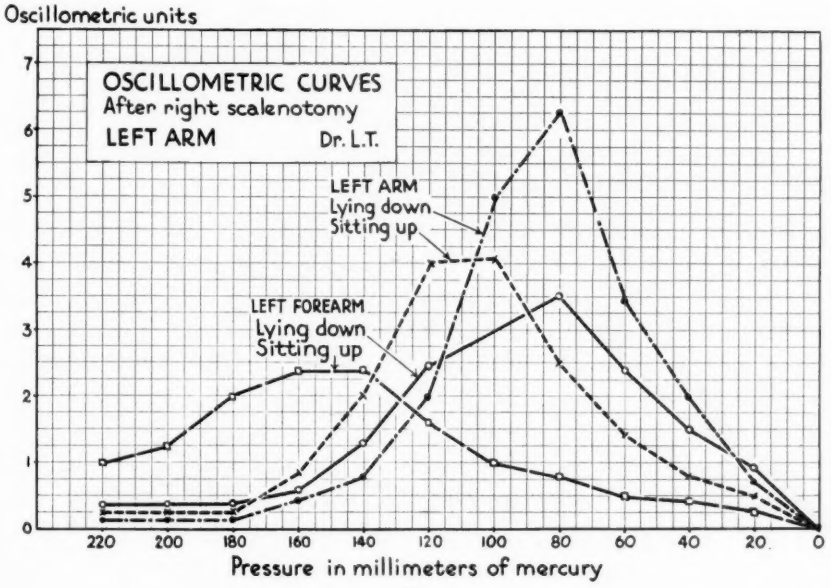
B.

Fig. 4.—Oscillometric curves of the right upper extremity before (*A*) and after (*B*) right scalenotomy (Case 2). In changing from the sitting to the reclining position before operation, the oscillometric index of the arm increased from 6.4 units to 9.9 units, while at the wrist the index increased from 2.5 units to 6 units. After operation the index of the arm increased from 4 units in the upright position to 6.3 units in the reclining position, while at the wrist the index increased from 2.4 units to 4.7 units. After operation the index in the reclining position was only at the level obtained in the sitting position before operation. If the changes in oscillometric readings were due to direct pressure on the subclavian artery, the same level should be reached in the reclining position both before and after operation. Loss of vascular tone which followed vasomotor irritation by the scalenus anticus muscle could account for these vascular changes.

The postoperative improvement in the ulnar nerve symptoms was associated with greatly reduced oscillometric indices compared to those observed before operation (Figs. 4 and 5). With direct pressure on the subclavian artery as the cause of peripheral circulatory changes, the same oscillometric readings should be obtained in the reclining position



A.



B.

Fig. 5.—Oscillometric curves of the *left* upper extremity on the same patient as charted in Fig. 4, before (*A*) and after (*B*) *right* scalenotomy. The index on the left extremity was reduced, although the operation was performed on the right side. This indicates that bilateral circulatory changes occur with unilateral operation. The average temperature of the fingers was always higher with the patient in the reclining position than in the sitting position.

both before and after operation. The greater index before operation can be explained by the loss of vascular tone due to the removal of the irritation or pressure on the vasomotor fibers of the brachial plexus when the patient assumes the reclining position. The absence or reduction of the irritation on the brachial plexus after operation is accompanied by less disturbance in vascular tone and consequently by a smaller variation in oscillometric indices in changing postural conditions.

CASE 3.—Mr. D. D., aged 41 years, a clerk, was referred Nov. 13, 1937, because of troublesome tingling and numbness of both hands and both arms but no circulatory symptoms or atrophy. There was some weakness of the hands but no objective sensory disturbances. At the onset of the above complaints, ten years previously, he had been examined and his condition diagnosed as a functional nervous disorder. Although the symptoms were bilateral, they were most prominent on the right side where digital pressure on the scalenus anticus muscle revealed definite tenderness. Similarly the right radial pulsation was reduced on deep inspiration when the shoulder was dropped, the head hyperextended and rotated to the right. This did not occur on the left side. In changing from the sitting to the reclining position, the oscillometric index increased 1.8 units in the right arm and 1.6 units in the right wrist. There was no change on the left side. Roentgenograms revealed large bilateral cervical ribs.

In spite of the indefiniteness of the neurologic symptoms, the physical findings of unilateral supraclavicular tenderness, and unilateral fluctuation in oscillometric readings and peripheral pulse as above described, right scalenotomy seemed to be warranted. The neurologist who had observed this patient for a number of years believed that removal of scalenus anticus pressure might benefit him.

On Nov. 24, 1937, right scalenotomy was performed and the cervical rib explored; no pressure being found, the rib was not disturbed. Both the muscle and posterior fibrous sheath were hypertrophied. Postoperative oscillometric readings showed that the index in the reclining position did not reach the level obtained prior to operation. This was also observed in the previous two cases, but the same unit increase occurred at a lower level in changing from the sitting to the reclining position than was noted before operation. The symptoms were not greatly relieved by the operative procedure.

Comment.—Symptoms suggesting a brachial neuritis with definite evidence of unilateral changes in the peripheral pulse and oscillometric readings seemed to indicate irritation or pressure on the brachial plexus. At operation the scalenus anticus was hypertrophied and was compressing the subclavian artery and brachial plexus. The cervical rib appeared merely incidental. This patient was not greatly benefited by scalenotomy.

DISCUSSION

The diagnosis of scalenus anticus syndrome is made primarily on the presence of clinical symptoms in the upper extremity which are influenced by posture. The symptoms are not clearly defined but pain, tingling, and numbness may occur anywhere from the shoulder to the fingers;[10] the usual distribution is along the course of the lower brachial trunks (ulnar or mixed median-ulnar nerve) with involvement of the

inner side of the arm, forearm, and the fourth and fifth fingers. Atrophy of the small muscles of the hand or even of the deltoid[11] and other muscles of the arm may occur. Circulatory changes, such as cyanosis, pallor, coldness, atrophy, or gangrene of the fingers, are less frequently observed than the neurologic symptoms; but recent reports,[12] as well as our observations, indicate that the disturbance is largely vasomotor in origin as a result of the brachial neuritis. Ochsner's observation of increased oscillometric index by injecting the cervicodorsal sympathetic nerves as well as our observations on certain characteristics of the oscillometric index (comment on Case 2 and Fig. 4) and on the lowered peripheral temperatures which are elevated to normal by novocain block of the peripheral nerves (Case 1) support the opinion that the syndrome is neurogenic. However, the subclavian artery[13] may be occluded by extravascular pressure or by thrombosis (Case 1).

Other pathologic conditions may be difficult to differentiate from scalenus anticus syndrome. These include inflammatory, traumatic, or toxic neuritis; subacromial or brachial bursitis; Raynaud's disease; ruptured supraspinatous tendon; and various cord, bone, and joint lesions. Further, mechanical irritation and pressure on the brachial nerves and subclavian artery can be produced by large cervical ribs or elongated transverse process of the cervical vertebra; by abnormalities of the first thoracic rib, brachial plexus, or subclavian artery; or by fibrous bands and adhesions in the supraclavicular triangle. It is obvious that even with the greatest care there will be difficulties in accurately diagnosing scalenus anticus syndrome and in establishing the cause of the brachial neuritis as due to compression by the scalenus anticus muscle.

Considerable importance has been given to diagnostic procedures for determining scalenus anticus pressure. The objective findings which are used to confirm the diagnosis are: (1) supraclavicular tenderness, especially to digital pressure on the trunks of the brachial nerves and scalenus anticus muscle; (2) aggravation of the peripheral symptoms by digital pressure on the scalenus muscle; (3) reduction or obliteration of the peripheral pulse with deep inspiration when the shoulder is dropped and the head is turned to the affected side and hyperextended; and (4) increased oscillometric index after novocain injection of the cervicodorsal sympathetic nerves (Ochsner). Since more than 50 per cent of the cases diagnosed as scalenus anticus syndrome give a history of trauma to the neck or shoulder, the tenderness and the aggravation of the peripheral pain by digital pressure could occur with a traumatic or any type of neuritis.

Oscillometric Readings.—To evaluate the importance of changes in the oscillometric index (maximum reading) as above described, a number of normal individuals were examined with a Pachon oscillometer (Table I). Instead of using Ochsner, Gage, and DeBakey's method of novocain block of the cervicodorsal sympathetic nerves, we took readings

TABLE I

NORMAL CONTROLS. INCREASE IN OSCILLOMETRIC READINGS OF ARM AND FOREARM IN CHANGING FROM THE SITTING TO THE RECLINING POSITION

PATIENT	AGE	SEX	OCCUPATION	MAXIMUM CHANGE OSCILLOMETRIC UNITS				SYMPTOMS
				ARM		FOREARM		
				R.	L.	R.	L.	
1. H. H.	30	F	Nurse	0.0	0.0	0.5	0.4	None
2. H. R.	31	M	Trimmer	2.4	2.8	0.7	1.2	None
3. A. P.	25	F	Secretary	0.0	0.4	0.6	0.4	None
4. F. B.	25	M	Student	2.4	1.8	0.8	0.8	None
5. J. R.	29	M	Student	0.0	0.4	1.3	1.2	None
6. H. W.	55	M	Executive	3.0	2.6	1.6	1.6	None
7. L. D.	32	F	Nurse	1.0	1.0	0.5	1.0	None
8. R. S.	44	M	Machinist	0.5	0.2	0.2	0.0	None
9. R. K.	29	M	Physician	2.0	2.0	0.7	1.0	None
10. N. B.	26	M	Student	0.5	1.5	0.7	1.2	None
11. A. N.	28	M	Technician	2.7	1.0	0.4	0.7	None
12. M. B.	25	F	Secretary	1.0	1.0	1.0	1.2	None
13. R. H.	25	M	Student	2.2	2.1	0.7	0.9	None
14. S. M.	57	M	Unemployed	0.0	0.6	0.2	0.0	None
15. R. A.	20	M	Student	1.0	0.9	0.5	0.7	None

with each change in posture and obtained a similar increase in oscillometric index. The majority of the normal controls had the same findings which have been used for confirming the diagnosis of scalenus anticus syndrome (Table II). The percentage of extremities with increased oscillometric index in the normal controls and of the involved and uninvolved extremities in suspected cases of scalenus anticus syndrome is contained in Table III. All of the involved extremities had some increase in the oscillometric index, while 25 per cent of the uninvolved extremities had no change. The larger increases also occurred more frequently in the involved than in the uninvolved extremity. Still, when compared to the group of normal controls, changes in oscillometric index must be cautiously accepted as a pathologic finding for confirming the diagnosis of scalenus anticus syndrome.

Peripheral Arterial Pulsations.—Reduction or obliteration of the radial pulse on deep inspiration when the shoulder is dropped and the head is turned to the affected side and hyperextended has been reported as significant of scalenus anticus syndrome. We found that a reduction or obliteration of the peripheral pulse occurs in most normal individuals. This was also noted by Spurling and Bradford.[8] These findings demonstrate that posture affects the peripheral pulse in almost all individuals, but the ease with which the pulse is reduced or obliterated may be the important consideration in the diagnosis of scalenus anticus syndrome.

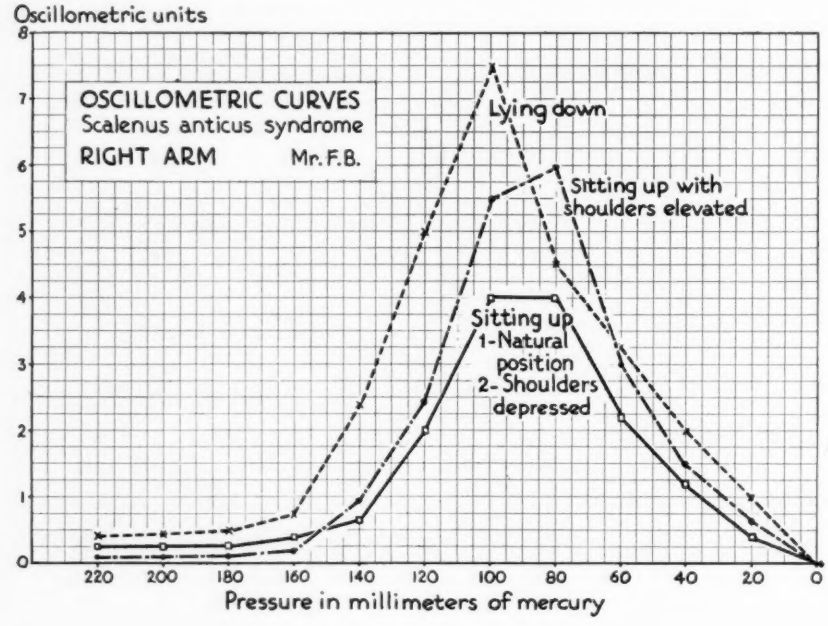
Treatment.—The fact that posture, stature, and muscular fatigue affect the severity of symptoms and the peripheral circulation (Fig. 6) indicates that conservative treatment is of definite value in alleviating the distress. Although Blair, Davies, and McKissock[12] studied histo-

TABLE II

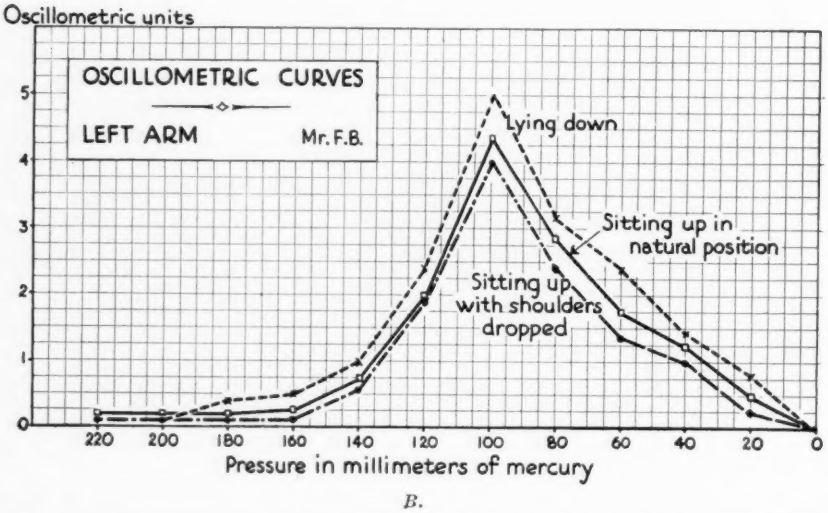
SUSPECTED SCALENUS ANTICUS SYNDROME. INCREASE IN OSCILLOMETRIC READINGS OF ARM AND FOREARM AND INCREASE IN AVERAGE PERIPHERAL TEMPERATURES OF FINGERS IN CHANGING FROM THE SITTING TO THE RECLINING POSITION

PATIENT	AGE	SEX	OCCUPATION	MAXIMUM CHANGE OSCILLOMETRIC UNITS				AVERAGE INCREASE IN TEMPERATURE		EXTREMITY INVOLVED
				ARM		FOREARM		FINGERS		
				R.	L.	R.	L.	R.	L.	
1. J. S.	36	M	Factory worker	0.4	0.4	0.2	0.2	+3.0° C.	+2.0° C.	Right
2. L. T.	47	M	Physician	3.5	1.5	3.6	2.2	+1.4° C.	+1.0° C.	Right
3. D. D.	42	M	Clerk	1.8	0.0	1.6	0.4	+0.2° C.	+0.0° C.	Right
4. F. R.	34	M	Telegrapher	0.5	0.4	1.0	1.0	+1.4° C.	+0.6° C.	Right
5. F. B.	33	M	Pianist	3.4	1.0	1.2	1.5	+0.4° C.	+0.7° C.	Bilateral
6. L. R.	55	M	Contractor	1.8	2.0	1.2	1.0	+0.5° C.	+0.5° C.	Right
7. S. J.	59	F	Housewife	2.5	3.4	1.5	0.6	+3.0° C.	+4.0° C.	Bilateral
8. R. P.	49	F	Housewife	0.5	2.5	1.8	1.8	-------	-------	Bilateral
9. E. D.	52	F	Housewife	2.0	1.8	1.6	0.2	-------	-------	Bilateral
10. V. C.	31	F	Housewife	1.6	1.8	0.4	1.5	-------	-------	Right
11. B. S.	38	F	Housewife	2.4	1.3	0.4	0.2	-------	-------	Right
12. B. C.	51	M	Lawyer	3.5	1.3	1.0	0.0	+1.5° C.	+1.0° C.	Left
13. C. E.	47	M	Contractor	0.0	3.0	1.0	1.0	+0.8° C.	+0.0° C.	Left
14. C. F.	40	M	Publisher	1.0	0.0	1.2	0.8	-------	-------	Right
15. J. H.	37	M	Physician	2.4	1.6	1.0	1.4	+1.0° C.	+1.0° C.	Right
16. M. D.	50	F	Housewife	2.5	0.5	0.8	0.0	+0.8° C.	+1.0° C.	Right
17. A. P.	43	M	Tailor	0.0	2.0	0.6	2.6	+1.0° C.	+1.2° C.	Left
18. T. W.	42	M	Factory worker	0.3	1.5	0.0	0.3	+0.7° C.	+0.6° C.	Left
19. W. S.	48	M	Machinist	1.5	2.0	1.9	1.5	+1.1° C.	+1.4° C.	Left
20. J. S.	39	M	Mechanic	2.0	2.0	0.0	1.3	+1.5° C.	+1.7° C.	Left

logically the brachial plexus in a case of cervical rib and found marked permanent changes in the nerves, routine operation is neither necessary nor is it always successful. Correction of posture, avoidance of fatigue, and in some instances immobilization of the arm and shoulder should be given a fair trial. When relief is not thus obtained, operative interference should be considered.



A.



B.

Fig. 6.—Oscillometric curves showing the effect of postural changes on the right and left extremities of a patient with right side scalenus anticus syndrome (*A*) (Case 3). On the left side (*B*) there is little change in the curve in assuming different positions, while on the right side the index increased from 4 units in the sitting position with the shoulders either depressed or in the natural position of the patient to 6 units with the shoulders elevated and to 7.6 units when lying down. Conservative treatment definitely benefited the patient.

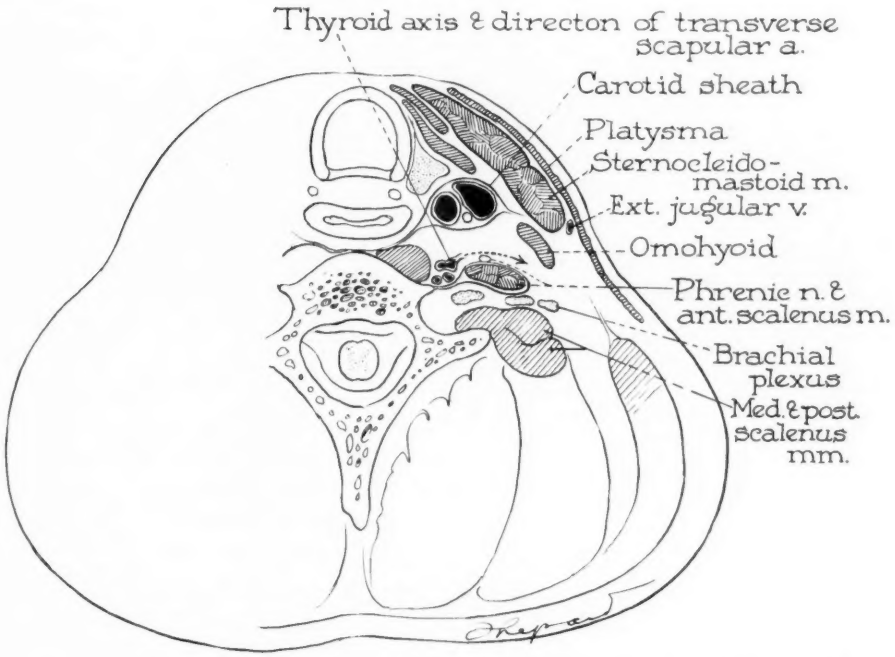


Fig. 7.—Transverse section of cervical region at level at which the scalenus anticus muscle is incised. The thick posterior fibrous sheath of the muscle should be divided with the muscle fibers. This portion of the muscle is directly over the cords of the brachial plexus and care should be taken that all the fibers are cut across.

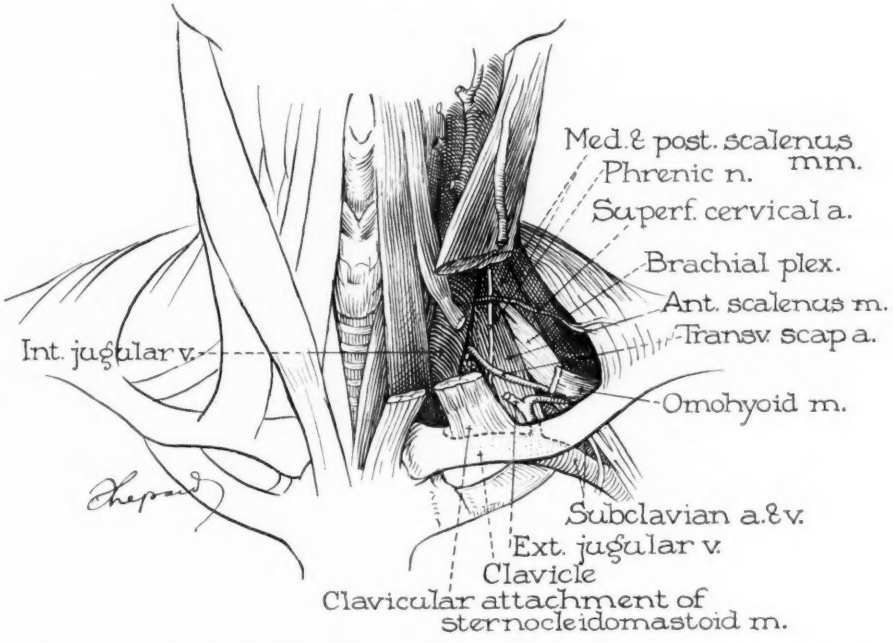


Fig. 8.—Showing the relationship of the scalenus anticus muscle to the brachial plexus and structures of the neck.

TABLE III

PERCENTAGE OF EXTREMITIES WITH INCREASED OSCILLOMETRIC READINGS IN CHANGING FROM THE SITTING TO THE RECLINING POSITION

	NUMBER OF EXTREMITIES	PERCENTAGE OF EXTREMITIES WITH INCREASED OSCILLOMETRIC INDEX				
		NO CHANGE	UNDER 1.0	OVER		
				1.0	2.0	3.0
Normal controls	30	16.6%	23.33%	60.0%	33.33%	3.33%
Suspected scalenus anticus syndrome						
Involved extremity	24	None	12.5 %	87.5%	54.16%	12.5 %
Uninvolved extremity	16	25.0%	25.0 %	50.0%	18.75%	6.25%

The operation of scalenotomy is not a difficult procedure. The scalenus anticus muscle (Fig. 7) is deeply placed and is in contact with the brachial plexus and the carotid sheath (Fig. 8). The thick fibrous posterior sheath of the muscle should be incised to permit maximum dropping of the first thoracic rib. Craig and Knepper[9] believe that elevation of the shoulder girdle due to spasm of the scalenus anticus muscle may produce the same symptoms as a cervical rib. Division of the hypertrophied muscle fibers alone will eliminate the pressure produced by spasm of the scalenus muscle. Unless the posterior sheath of the muscle is divided, the first rib may continue to be elevated and to produce pressure on the nerve trunks and artery.

CONCLUSIONS

Scalenus anticus syndrome is a symptom complex which is due to compression of the brachial plexus. All suspected cases must be carefully scrutinized for the presence of other neurologic conditions.

The diagnostic procedures which demonstrate changes in the peripheral pulse volume and oscillometric index due to posture are not pathognomonic of the disease. The same findings may occur in normal individuals as well as in suspected cases of scalenus anticus syndrome.

Scalenotomy is not indicated in every case of scalenus anticus syndrome. Conservative treatment will benefit most cases with complete relief of symptoms. The best operative result is obtained when a cervical rib is present.

REFERENCES

1. Editorial: Cervical Rib and Scalenus Anticus Syndrome, J. A. M. A. **109**: 877, 878, 1937.
Naffziger, H. C.: Editorial, The Scalenus Syndrome, Surg., Gynec. & Obst. **64**: 119, 120, 1937.
2. Naffziger, H. C., and Grant, W. T.: Neuritis of the Brachial Plexus Mechanical in Origin; the Scalenus Syndrome, Surg., Gynec. & Obst. **67**: 722-730, 1938
3. Murphy, J. B.: A Case of Cervical Rib With Symptoms Resembling Subclavian Aneurysm, Ann. Surg. **41**: 399-406, 1905.
4. Adson, A. W., and Coffey, J. R.: Cervical Rib, Ann. Surg. **85**: 839-857, 1927.
Adson, A. W., and Coffey, J. R.: Cervical Rib, Method of Anterior Approach for Relief of Symptoms by Division of Scalenus Anticus, Ann. Surg. **85**: 839-857, 1937.

5. Naffziger, H. C.: Quoted by Ochsner.[7]
6. Boland, E. R.: Cervical Rib With Symptoms Due to Dropping of the Shoulder Caused by Obliterative Pleurisy, Brit. M. J. **2:** 1068, 1069, 1933.
7. Ochsner, A., Gage, M., and DeBakey, M.: Scalenus Anticus (Naffziger) Syndrome, Am. J. Surg. **28:** 669-695, 1935.
8. Spurling, R. G., and Bradford, F. K.: Scalenus Neurocirculatory Compression, Ann. Surg. **107:** 708-715, 1938.
9. Adson, A. W.: Surgical Treatment of Cervical Ribs, Texas State J. Med. **28:** 739-747, 1933.
Kasman, L. P., and Berstein, W.: Cervical Rib, Am. J. Surg. **30:** 372-375, 1935.
Craig, W. McK., and Knepper, P. A.: Cervical Rib and the Scalenus Anticus Syndrome, Ann. Surg. **105:** 556-563, 1937.
Bertelsman, R.: So-Called Scalenus Anticus Syndrome; Therapy by Scalenotomy, Chirurg. **9:** 464, 465, 1937.
10. Flothow, P. G.: Cervical Rib and Anterior Scalenus Syndrome, West. J. Surg. **44:** 570-574, 1936.
Wanke, R.: Scalenus Syndrome and Cervical Rib, Arch. f. klin. Chir. **189:** 513-528, 1937.
11. Ochsner, H.: Footnote 7.
Wanke, R.: Footnote 10.
12. Blair, D. M., Davies, F., and McKissock, W.: Etiology of Vascular Symptoms of Cervical Rib, Brit. J. Surg. **22:** 406-414, 1935.
Telford, E. D., and Stopford, S. B.: The Vascular Complications of Cervical Rib, Brit. J. Surg. **18:** 557-564, 1931.
Langeron, L.: Vascular Disturbances of Upper Extremities Caused by Cervical Ribs, Paris méd. **2:** 43-45, 1933.
Leriche, R.: Late Results of Surgical Therapy of Cervical Rib, Bull. et mém. Soc. nat. de chir. **61:** 1292-1300, 1935.
Spurling, R. G., and Bradford, F. K.: Footnote 8.
Newell, R. L.: Cervical Rib With Vascular Complications, Brit. M. J. **1:** 782, 783, 1933.
13. Robinson, S., Stone, C. S., Jr., and Elliot, A. J.: Cervical Ribs, West. J. Surg. **43:** 295-304, 1935.
14. Robinson, S., Stone, C. S., Jr., and Eliot, A. H.: Footnote 13.
Singleton, A. O.: Discussion, Am. J. Surg. **28:** 669-695, 1935.
Brickner, W. M., and Milch, Henry: First Dorsal, Simulating Cervical Rib—By Maldevelopment or by Pressure Symptoms, Surg., Gynec., & Obst. **40:** 38-44, 1925.

A SIMPLE TECHNIQUE FOR CHOLECYSTGASTROSTOMY

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THE rationale for short-circuiting procedures on the biliary tract, such as cholecystgastrostomy or cholecystduodenostomy, is an irremovable obstruction to the common bile duct. Such an obstruction is due most commonly to carcinoma of the head of the pancreas or the ampulla of Vater and occasionally to fibrotic stenoses. Whereas cholecystduodenostomy may appear the operation of choice because bile empties normally into the duodenum, it usually cannot be performed as readily as cholecystgastrostomy because of the frequent inaccessibility of the duodenum. Moreover, cholecystgastrostomy apparently functions satisfactorily.

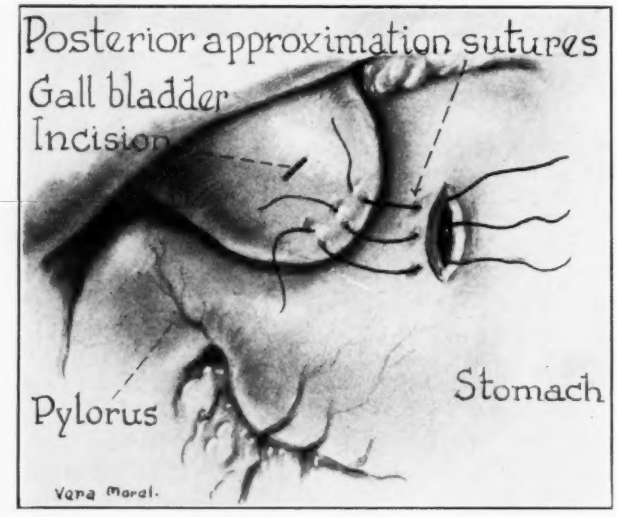

Fig. 1.—Drawing showing posterior row of approximation sutures between lateral edge of incision in stomach wall and fundus of gall bladder.

The technique usually described for the performance of cholecystgastrostomy consists of a suture anastomosis similar to the classical gastrointestinal anastomosis. In 1930 the late J. Tate Mason[1] described a much simpler procedure which has the added advantage of a certain amount of muscular control at the site of the stoma. The procedure consists briefly of introducing a curved hemostat into the lumen of the stomach through a small incision in its anterior wall near the pylorus and gently pushing the blades through the stomach wall

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a few centimeters above and distal to the initial incision. The fundus of the gall bladder is then caught with the hemostat and pulled into the lumen of the stomach through the second gastric opening and fixed in this position with a single row of silk sutures. An opening is made in the fundus of the gall bladder by means of a pair of scissors introduced through the initial incision which is subsequently closed.

We have performed the procedure as originally described and found

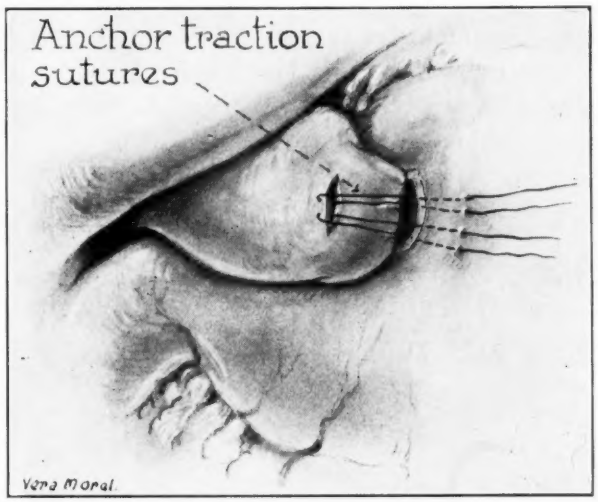

Fig. 2.—Posterior row of approximation sutures have been tied and severed. Anchor traction sutures are simply mattress sutures of silk with the original point of entry and the final point of exit opposite each other in the anterior stomach wall about 1 cm. from the gastric incision, through which they pass to be applied in the upper edge of the incision in the fundus of the gall bladder.

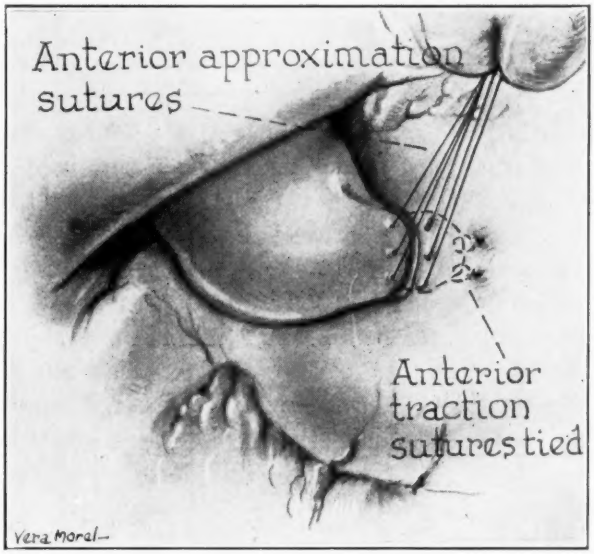

Fig. 3.—Anchor traction sutures have been tightened and tied, drawing that portion of gall-bladder fundus in which opening is made through incision into lumen of stomach as represented by the dotted line. The anterior row of approximation sutures is placed between edge of gastric incision and adjoining wall of gall-bladder fundus.

it to function quite satisfactorily. More recently, however, a slight modification has been employed which is believed further to simplify and facilitate its performance.

The technique consists of making a small incision, 1 to 1.5 cm. in length, in the anterior stomach wall near the pylorus and the lesser curvature. Because the gall bladder is usually distended in these cases, the preliminary aspiration of its contents will greatly facilitate its manipulation. A posterior row of approximation sutures are then placed between the fundus of the gall bladder and the lateral edge of the incision in the stomach (Fig. 1). Three or four interrupted fine silk sutures usually suffice. A small incision, about 6 to 7 mm. in

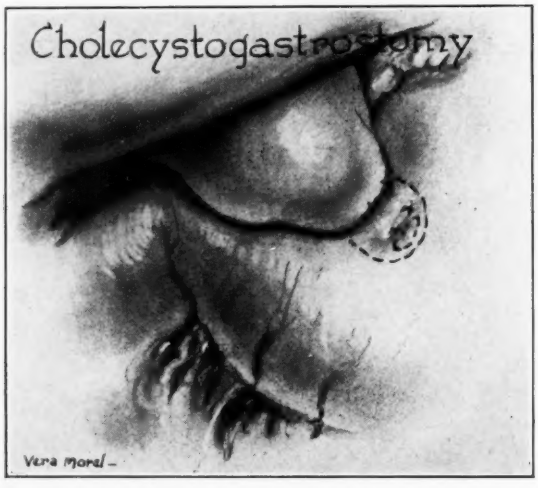

Fig. 4.—Anterior row of approximation sutures has been tied and severed completing the cholecystgastrostomy procedure. A small omental graft may be placed over suture line for reinforcement.

length, is made in the fundus of the gall bladder about 1 cm. from and parallel to the posterior approximation suture line (Fig. 1). Anchor traction sutures are then inserted as illustrated in Fig. 2. Traction upon these sutures draws that portion of the fundus of the gall bladder in which the opening is made through the incision into the lumen of the stomach (Fig. 3). The tying of these sutures helps to secure this position. An anterior row of approximation sutures is then placed between the edge of the gastric incision and the adjoining wall of the gall-bladder fundus (Fig. 3). These three or four interrupted silk sutures are tied, completing the operation (Fig. 4).

REFERENCE

1. Mason, J. Tate: Technic of Cholecystogastrostomy, J. A. M. A. **94**: 29, 1930.

Editorial

Thrombophlebitis

THROMBOPHLEBITIS and its sequelae are of great importance to the clinician because of the occasional tragic outcome and because of the prolonged convalescence following a thrombophlebitic process. In considering intravascular clotting, it is important to distinguish between those lesions which are associated with an inflammatory process in the vessel wall, a true thrombophlebitis, and those in which there is intravascular clotting without the associated inflammatory lesion which can be termed a "phlebothrombosis." It is the latter type which is particularly dangerous, because patients having this condition have relatively few symptoms referable to their vascular systems and because of the absence of inflammatory reaction which tends to fix the thrombus to the vessel wall the likelihood of detachment of the clot and the production of a fatal pulmonary embolism are particularly great.

There are many factors which predispose to the development of thrombi. Trauma of any kind, whether it is operative or is the result of destruction of tissue from infection, predisposes to thrombosis by altering the constituents of the blood. These alterations which favor intravascular clotting are increase in number of thrombocytes and increased agglutinability of the corpuscular elements of the blood. Operative procedures also predispose to thrombosis because of circulatory retardation, the result of relative immobility, postoperatively particularly in the lower extremity. Similarly, circulatory retardation is favored by vasospasm of the peripheral vessels which usually occurs postoperatively. The diminished respiratory activity following an abdominal operation also favors circulatory retardation because of (1) the decreased intrathoracic negative pressure which normally aids in the return of the blood to the thorax, and because of (2) increased abdominal tension resulting from the use of tight abdominal bandages or gastrointestinal distention which compresses the intra-abdominal veins and favors stasis in the lower extremity. Pelvic operations both in the male and female are more likely to be associated with postoperative thromboses than operations elsewhere.

Most patients with thrombophlebitis or phlebothrombosis have some symptoms referable to the involved veins. As emphasized by Homans, the sites involved are most likely to be those areas usually below the entrance of many tributaries. In the lower extremity these are usually in the femoral iliac region and in the calf of the leg just below the popliteal area. The occurrence of pain in the leg associated with fever

and tenderness limited to and over the course of a vein is of diagnostic importance. The pain is characteristically increased by having the extremity in a dependent position and is relieved somewhat with the extremity elevated. In most instances there is edema. In thrombophlebitis of the deep veins there is almost invariably a decrease in the peripheral blood supply. Because of this, the name phlegmasia alba dolens has been given to the condition. It is because of the diminished peripheral blood supply and its decreased radiation of heat that the name alba, or white, has been used. However, until relatively recently, little significance has been attached to diminished peripheral blood supply.

It is a well-known fact that in most cases of phlegmasia alba dolens, which are usually the result of femoral-iliac thrombophlebitis and which are frequently associated with postpuerperal or postoperative infections, the prognosis is not good. These patients have fever, marked swelling of the involved extremity due to the edema, and tenderness over the course of the deep vein. These symptoms usually persist for from four to six weeks. Generally, the patients are kept quietly in bed for this period of time and for an additional two to three weeks after the subsidence of the fever, following which they are allowed up. As a rule, after the patient is up there is a return of the edema which is likely to persist for months, even years, and not infrequently for the rest of the life of the individual. Relatively little has been accomplished in the treatment of such cases.

With the introduction of a purified heparin by Best and his associates, much can be accomplished in the prevention of postoperative thrombosis in patients who are potential clotters. The detection of such individuals by the Bancroft formula and the intravenous infusions of heparin over a period of time postoperatively may accomplish a great deal. However, because heparin has relatively little effect upon the condition after a coagulum has occurred, it has limited use in cases of thrombophlebitis or phlebothrombosis in which thrombosis already has occurred.

Leriche first called attention to the beneficial effects of novocain block of the sympathetic ganglia in cases of thrombophlebitis. Based upon our own experience, we are convinced that this is the most efficacious method of treating thrombophlebitis because it results in the re-establishment of normal vascularity within a very short period of time. As a result of recent clinical and experimental observations, we are convinced that an entirely different conception of the development of edema associated with thrombophlebitis is necessary. Contrary to the generally accepted theory that edema of thrombophlebitis is due to an increased venous pressure resulting from blockage of the deep veins, by the occluding thrombi, we believe that, whereas, this may play some role, vasospasm is a far more important factor. According to Leriche, the edema is the result of venous stasis caused by reflex venospasm. We are of the opinion, however, that the decrease of the arterial supply with its

resultant relative tissue anoxia and the diminution in arterial pulsation are the principal factors in the production of edema in thrombophlebitis. We have demonstrated that in thrombophlebitis there is marked diminution in the peripheral arteriolar pulsation in the involved extremity resulting probably from vasospastic influences originating in the thrombophlebitic process. As a result of the decreased blood supply and relative ischemia (anoxia) of the capillary endothelium occasioned by the diminished blood supply, there is an increased permeability of capillary endothelium resulting in an increased exudation of plasma into the perivascular spaces. The persistence of the edema is facilitated by the fact that, if the fluid once gets into the perivascular spaces, it remains there because of the absence of pulsation. Investigations by McMasters have showed that the movement of the lymph is, to a great extent, if not wholly, dependent upon the arteriolar pulsations. In the absence of such pulsations it is probable that the perivascular fluid remains and is not carried away, which accounts for the persistence of the edema. Unless the fluid is removed within a relatively short period of time, that is, within two to three weeks, there is the likelihood of the edema persisting because of fibrosis, which, as is well known, is favored by lymph stasis. It is probably for this reason that the patients who have a phlegmasia alba dolens develop a persistent edema which lasts the rest of their life.

By overcoming the vasospasm in patients with thrombophlebitis by repeated injections of the lumbar sympathetic ganglion with novocain, it is possible to bring about a restitution of normal vascularity within a very short period of time. There is a complete relief of pain within one-half hour after injection; the temperature rapidly falls to normal; and there is a complete subsidence of the swelling of the extremity within two weeks' time. By preventing the persistence of edema, subsequent fibrosis with persistence of symptoms is obviated.

—*Alton Ochsner.*

Recent Advances in Surgery

Conducted by Alfred Blalock, M.D.

RECENT ADVANCES IN THE MORE COMMON PROBLEMS OF MINOR ANORECTAL SURGERY

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(Continued from the June issue)

CRYPTITIS

A conspicuous advance in the treatment of anorectal disease has been increasing recognition of the important role played by infection of the crypts of Morgagni in the development of fissure-in-ano, perianal and perirectal abscesses, and fistula-in-ano. This will be discussed further under these headings. Recognition of crypt infection is dependent upon careful anoscopic examination. Proper evaluation of its significance depends upon the experience and judgment of the examiner. Deformity of the crypt margins, enlargement and irregularities of the anal papillae as the end result of an infection long since played out may be of no consequence. The finding of an abnormally deep crypt by exploration with a hooked probe does not by itself warrant the diagnosis of cryptitis. Active infection is revealed by the presence of true signs of inflammation: redness and possibly edema of the margins of the crypt, redness and ulceration of the mucous membrane immediately above the mouth of the crypt, extreme sensitivity to gently probing, and frequently edematous enlargement of the adjacent papillae. It is sometimes possible by pressure over a crypt to express a drop or two of actual pus.

Recognition and treatment of cryptitis in its early localized stage are of great importance in the prevention of numerous complications, notably fissure, abscess, fistula, and anal stenosis. Buie[28] regards crypt infection as an important etiologic factor in the development of hemorrhoids.

The usual treatment consists in the application of local heat (hot Sitz baths), lubrication of the stool, and direct application of some medicant. It has been emphasized that the value of the latter depends upon its application with the lower rectum empty of bowel discharges. This can be assured by office procedure or by the employment by the patient of local irrigation with a small warm enema of plain water or saline solution. Some of the drugs utilized include aqueous solution

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of metaphen,[28] witch hazel,[28] aqueous solution of ichthyol 25 per cent,[7] pure ichthyol,[193] protargol, 1 per cent,[7] and pyridium, 0.5 per cent.[7] Drueck[46] flushes each diseased crypt thoroughly with water by means of a syringe with an angulated nozzle and generously applies 2 per cent mercurochrome or pure ichthyol for more active stimulation. When such daily treatment is impractical, I have found efficacious the self-application of 15 per cent ichthyol in lanolin by means of a collapsible tube with perforated tip attachment after bowel movements and shortly before retiring.

Operative procedures should be reserved for deep, obviously infected crypts which fail to respond to nonsurgical measures. Simple local excision of the crypt under local infiltration anesthesia has been employed,[7, 193] but the importance of exterior drainage of the wound well outside the anus has been emphasized by Hirschman[95] and others[28, 56] to insure uncomplicated healing. Diathermy has been recommended for cryptectomy by some.[24, 64, 143] Surgery in the presence of a gonorrheal cryptitis is inadvisable.[28]

Considerable importance has been placed upon the role of cryptitis as a focus of infection in various systemic affections by a few.[94, 96, 134, 143] I have seen several patients with persistent *B. coli* infection of the urinary tract which was unrelieved until a gross infection of an anterior anal crypt with a small associated abscess was eradicated. That such infection may be a possible seat for so-called focal infection cannot be denied. But its overemphasis as the explanation of a great variety of constitutional and neurotic disorders is greatly to be deplored.

The importance of the anal ducts, tubular glands emptying into the depths of the anal crypts, in infections in the region of the crypts is emphasized by Tucker and Hellwig,[182] who have made a careful study of their histology and embryonic development. Earlier mention of the possible relationship of these vestigial glands to the development of fistula-in-ano was made by Herrmann and Desfosses[86] in 1880 and later by Harris[79] and Lockhart-Mummery.[108]

Hypertrophy of Anal Papillae, Papillitis.—Edema, inflammation, and hypertrophy of the anal papillae are secondary to anal cryptitis. The recognition of this fact is important in diagnosis and treatment. Frequently a progressive fibroblastic hypertrophy of a papilla may be incited by chronic crypt inflammation and may continue even after the active crypt infection has subsided. Such a condition may become the source of a definite annoyance to the patient and may require surgical excision. Simultaneous excision of the adjacent crypt is important in the prevention of a recurrence of the condition. In the excision of such an hypertrophied papilla Buie[28] recommends approximation of the skin edges at the upper end of the incision by a single suture. This would appear inadvisable when simultaneous excision of a crypt is performed.

CONTRACTURE OF THE ANAL CANAL

Any local, painful condition in the region of the anal canal results in an associated reflex spasm of the sphincters with ultimately considerable hypertrophy of the anal musculature. It is important to realize that a tight, unrelaxing anus may be entirely secondary to such primary disease and may be entirely relieved by correction of this condition. Anal spasm occasionally may be the result of prolonged cathartic or enema habituation and very rarely may develop secondary to irritative conditions of the lateral tracts of the spinal cord.[28]

In contradistinction to purely spastic contracture there does occur a condition in which there is a true inelastic fibrous constriction of the upper end of the anal canal. Over a hundred years ago the occurrence of such a condition was noted and was considered to be the result of spasm of the sphincter muscles.[121] In 1919 Miles[126] described a subcutaneous circular fibrous band which he termed the "pecten band" declaring that this condition was the result of chronic passive congestion. Abel[1] in 1932 agreed that this condition was due to chronic passive congestion and further declared that the continued spasm of the anal sphincters will cause passive congestion and subsequent fibrosis. He called the condition "pectenosis." Although there has been general recognition of the occurrence of such fibrotic constrictions, the theory of chronic passive congestion as the underlying cause has not received wide recognition in this country. To many this deposit of fibrous tissue has appeared rather to be due to the result of chronic infection arising in the anal crypts. This theory has been expounded in an excellent article on the subject by Buie.[29] This authority characterizes the condition as an inelastic fibrous stricture of the anus, including subcutaneous and muscular components, due to infection which gains entrance through the anal crypts, usually with accompanying pathologic changes. Anal contracture on the basis of congenital stenosis has been described in children[185] and in adults.[90]

Division of the pecten band by posterior midline incision was termed "pectenotomy" by Abel.[1] Although this operation is employed by some today an inherent weakness exists in the lack of recognition of the true etiologic factor concerned. Permanent correction of the deformity must include removal of any existing source of continued infection, the most frequent of which is a chronic fissure-in-ano or a chronically infected crypt. Buie[29] stresses the correction of any accompanying pathologic deformity and advocates careful intelligent stretching to overcome the contractural influence of fibrous deposits rather than incision.

FISSURE-IN-ANO

Differentiation must be made between superficial cracks in the skin, single or multiple, and the chronic, usually single fissure which extends through the entire thickness of the anal skin into the underlying areolar tissue. The former may result from external irritation, the irritation of some form of rectal discharge, or from simple mechanical splitting of the anal skin by a large, hard stool. These "acute" fissures heal readily with almost any type of treatment or often with no treatment at all; whereas, the permanent cure of a "chronic" fissure may tax the resources of the attending physician.

The pathogenicity of chronic anal fissure is still subject to dispute. Yet upon its determination rests the rationale of proper treatment. The time-honored theory of Ball[10] that a fissure is the result of a tearing down of an anal valve by a scybalous mass is still accepted by some. Others[73, 83] support the theory of simple traumatic splitting of the anal skin by a hard, dry stool, as advocated by Brodie[25] over a hundred years ago. Riddoch[149] has proposed the theory that a small extravasation of blood subcutaneously as the result of the trauma of a hard stool leads to a ridge of fibrous tissue which sooner or later ruptures with the development of a fissure.

Lockhart-Mummery[109] called attention to the V-shaped deficiency in the sphincter muscle posteriorly where the muscle fibers, diverging from the coccygeal attachment to pass on either side of the anal canal, are a site of weakness. He considered this to be the causative factor in fissure-in-ano. In 1934 Milligan and Morgan[128] published an elaborate essay describing three zones of the external sphincter muscle: an inner (deep) and outer (subcutaneous) portion consisting of circular bands of muscle without bony or ligamentous attachment, and a middle (superficial) portion with attachment to the coccyx and anococcygeal raphé and a divergence of fibers just posterior to the anus. Blaisdell[18] explains further that the outer or subcutaneous portion of the sphincter forms a "bar" type of interference that causes the skin within its grasp to be broken during the act of defecation. This authority considers this of great importance in etiology and treatment.

Hiller[92] believes that firm fixation of the posterior commissure and to a lesser extent the anterior commissure by fibroelastic termination of the longitudinal bowel coat plus lateral free mobility of the mucous coat probably plays a greater role than does the contour of the external sphincter. Haws[83] stresses the strong protection furnished the lateral walls of the rectum by the levator ani muscles leaving two weak areas at the anterior and posterior commissures.

The high incidence of posterior fissure-in-ano is in large measure attributed[28] to the peculiar relationship of the anal walls to those of the rectum. Whereas the anterior anal wall joins with the rectum in

a plane which is more or less flat, the posterior anal and rectal walls meet at right angles. Moreover, because the rectal ampulla curves around in the hollow of the sacrum, the descending fecal mass thrusts against the posterior wall and practically the entire expulsive force is exerted on the posterior rectal wall just above the posterior anal margin, on that portion of the anal wall with the least support.

With due appreciation of the importance of anatomic structure in the occurrence of fissure-in-ano, it is nevertheless being increasingly recognized that infection originating in an anal crypt is a primary factor in its development. Mounsey[134] recognizes crypt infection as the starting point of fissure-in-ano in 20 per cent of cases. Speare and Mabrey[173] consider it important in the pathogenesis. Fansler[50] describes a downward superficial spread of infection from a crypt rendering the "mucosa" friable with subsequent splitting caused by slight traumatization. Bacon[7] refers to cryptitis as a usually "associated condition." Buie[28] considers that infection originating in a crypt is the primary cause, with trauma the inciting factor. I believe that recognition of infection in an anal crypt or an anal duct as the underlying cause of chronic anal fissure is of fundamental importance in the treatment of this condition. An inflammatory process spreading downward from a crypt renders friable the overlying anal skin, causing splitting by the ordinary trauma of defecation. This split often does not occur directly over the crypt but distal to it. It also frequently does not reach to the outermost limits of the inflammatory spread so that a pocket may develop at the distal end of the fissure. This pocket and the continued reinfection from the superimposed crypt are two important reasons for the chronicity of the condition. The mechanical factors enumerated above are undoubtedly significant in the explanation of the high percentage of fissures occurring in the posterior midline. But it has never been shown that patients who have a fissure-in-ano have been subjected to greater defecatory trauma than countless individuals who never develop such a fissure.

Treatment of Anal Fissure.—In discussion of the treatment of anal fissure, immediate differentiation must be made between the acute and the chronic fissure. An acute fissure is frequently the result of a simple mechanical splitting of the anal skin and may be healed by many types of simple local treatment or may heal with no treatment at all. Many of the cures by nonsurgical methods enthusiastically reported today owe their success in part at least to their effectiveness in this type of fissure. It is my firm conviction that with greater recognition of the theory that every chronic fissure-in-ano is the result of a downward spread of subcutaneous infection from an anal crypt, uniformly satisfactory and rational treatment will ensue. Some types of procedure, advocated consciously or unconsciously, satisfactorily effect rational cure on the basis of this theory of etiology. But emphasis

upon the role of such infection is conspicuously lacking in the literature. Again, many of the nonsurgical methods of treatment may effect a cure in a certain percentage of even this type of chronic anal fissure. Healing is not permanent, however, unless, happily, the responsible infection of the superimposed anal crypt or duct has spontaneously subsided. This does not mean that there is no proper place for these methods of treatment. But it is a plea for their intelligent and rational use in a condition which is a cause of great suffering and from which the patient so ardently desires early, lasting, and permanent relief. The presence of certain features that can be determined by proper examination are definite indications for what might be considered by some radical surgical interference. These features include the presence of a burrowing pocket of infection distal to the fissure, with or without a so-called sentinel pile; the existence of an obviously chronically infected superimposed crypt; and the presence of a definite bar of subcutaneous fibrosis at the upper end of the anal canal, in itself evidence of a chronic inflammatory process secondary to infection by way of an anal crypt. Ideal operative treatment in the presence of any of these above named conditions requires hospitalization for from two to four days. If proper radical operation can be done and control of postoperative discomfort can be obtained by the use of suitable anesthetic agents of prolonged action as an office procedure, this method of treatment is justifiable and commendable. There is usually, however, an almost inevitable tendency to temporize and to minimize any surgical procedure when the treatment of anal fissure is considered routinely an office procedure. There are also certain objections to the employment of the various agents for prolonged anesthesia which have already been discussed. Their true value will be learned only by further investigation and use. In the absence of evidence of the above criteria for operative interference nonsurgical methods of treatment are worthy of trial. If such treatment fails, radical operation should be employed here also.

It has frequently been considered that the cardinal principle in treatment of anal fissure is rest. For this purpose divulsion of the sphincter was employed. While still used by some,[7, 109, 193] it has been increasingly recognized that such a procedure is illogical and undesirable. Buie[28] considers that this practice, along with cauterization operation for hemorrhoids, poorly conceived diagnostic methods, and either poor postoperative management or none at all, has retarded the advance of anorectal surgery a full generation. More recently employed for the purpose of rest have been various agents for prolonged anesthesia, both in aqueous and in oily solutions. These agents have already been discussed under anesthesia and need not be reviewed here. From 3 to 10 c.c. of such a solution is injected beneath the fissure and into the sphincter on either side through a single needle punc-

ture. Favorable reports on this method of treatment of anal fissure have been numerous in the literature.[67, 73, 83, 104, 149, 173, 187]

Operative Procedures.—Numerous methods of surgical treatment are employed: incision through the ulcer carried out onto the perianal skin, with or without division of a variable amount of the external sphincter muscle; conservative excision of the margins of the fissure with little if any emphasis placed upon adequate external drainage; and more radical, complete excision of the entire fissure and its bed with triangular excision of skin distally to obtain suitable external drainage. Gabriel[56] has emphasized the necessity of triangular excision, the apex of the triangle at the mucocutaneous line, the base well outside the anal verge so that healing will occur from above down, the last area to heal being outside the anus. This sound principle has been utilized by others.[7, 28, 50, 134, 172] The triangular excision should include the superimposed crypt. Buie's operation[28] adequately does this. The routine suture of the free edge of mucous membrane to the sphincter is supposed to facilitate prompt healing. Excision should include all fibrous tissue down to the surface of the sphincter muscle. If any contracture is noted as the result of deeper fibrosis in the sphincter itself, this may be incised or gently dilated. Blaisdell[18] recommends incision of the bar formed by the circular fibers of the subcutaneous portion of the external sphincter muscle. This may well be done to assure perfect distal drainage. By operation based upon the methods of Gabriel, Buie, and Blaisdell it is possible to remove the primarily infected anal crypt, the distal pocket of subcutaneous infection, and the constricting band of fibrous tissue which may be present, with provision for correct healing of the wound from above downward. Such a procedure will be found most satisfactory in the permanent cure of anal fissure. Careful postoperative care is essential to uniformly correct healing. This is similar to that described for hemorrhoidectomy. The cardinal object of such treatment is maintenance at all times of adequate distal drainage.

ANAL ABSCESS AND ANAL FISTULA

Pathogenesis.—Anal abscess and anal fistula represent successive stages in a common infectious process and may well be considered together in discussion of their pathogenicity. Operation for fistula-in-ano is second only to trephining as the oldest procedure known to surgery.[108] The many recurrences and the many tragedies of permanent impairment of sphincter control that still occur following operation bear witness to the fact that even today essential knowledge of pathogenesis and proper procedure is not widespread.

Certain desirable changes and interpretations in nomenclature have been suggested by Buie.[27, 28] "Anal fistula" and "rectal fistula" are not synonymous. A rectal fistula develops as the result of destruction

of the continuity of the rectal wall by granulomatous or neoplastic disease, the fistula being secondary to one or another primary specific disease of the rectum. An anal fistula is the result of infection originating always in the region of the anal crypts. The fistulous opening at the point of origin Buie terms the "primary" opening and the terminal opening he designates as "secondary." This terminology accords with the facts and eliminates the confusion which may occur when the secondary opening, formerly called the external opening, happens to be internal.

Among the various etiologic factors listed by many of the textbooks on anorectal disease are irritation from clothing or harsh detergents; infections in sebaceous glands and hair follicles; abrasions, tears or wounds of the anal or perianal skin or of the rectal mucosa; infection of internal or external hemorrhoids; rectal or anal ulcers; infection primary in the prostate, seminal vesicles, urethra, broad ligament and appendix; and lymphatic- and blood-born infections. Some of these books mention the possibility of primary infection in the anal crypts; others do not. However, recognition of the importance of these crypts as the primary site of infection has been gradually increasing in the past decade.[6, 15, 39, 167, 177] The ultimate recognition of this fact is expressed by Buie[28] in the assertion that all abscesses of anal origin come from a source within the anal crypts and that the primary (internal) opening of every fistula-in-ano is always within the crypts of Morgagni. Trauma may be an inciting cause of the original crypt infection. Lockhart-Mummery[108, 110] and others[79, 182] have called attention to the anal ducts or vestigial "intramuscular glands" as the frequent site of origin for anal abscesses and fistula. These glands, opening into the base of the anal crypts, may extend a variable distance through the sphincter muscles and at times may actually reach the ischiorectal fossa. I consider these anal ducts of great etiologic significance in the development of anal abscess and fistula in infants. Although these are commonly considered rare in the newborn, they are of fairly frequent occurrence in my experience, having three such infants under 4 months of age in my care at the present time. Two of these patients had two separate complete fistulas which had persisted following the development of anal abscesses within the first two or three weeks of life. Histologic examination revealed a glandular epithelial lining in portions of the fistulous tracts.

The role of tuberculosis in the development of anal abscess and fistula has long been a subject of much discussion. The incidence of tuberculous fistulas reported in the literature varies from 1.4 per cent to 61 per cent.[31] A wide variation (4 to 100 per cent) is noted in the incidence of tuberculous fistulas among patients with pulmonary tuberculosis.[31] These extremely divergent reports are in large part dependent upon the criteria of diagnosis. Diagnosis by characteristic

appearance of the fistula is considered by most authorities to be unreliable. Others[117, 161] claim that they are easily distinguishable. Diagnosis from the histologic appearance of the tissue is considered unsatisfactory. Gabriel[55] has shown that demonstration of the tubercle bacilli in the tissues is difficult and unreliable. This authority believes that accurate diagnosis is best made by animal inoculation. Magath and Feldman[114] have found that guinea pig inoculation is more reliable than cultural methods. Chisholm and Gauss,[38] after extensive study of the literature, concluded that probably from 3 to 5 per cent of anal fistulas are tuberculous. After careful study by inoculation methods, Gabriel[55] believes that about 15 per cent are tuberculous. In a recent study of over 200 consecutive cases by intraperitoneal and subcutaneous inoculation of guinea pigs, Buie and co-workers[31] obtained positive results in 11.3 per cent of cases, although microscopic study of many sections of tissue proved the presence of tuberculosis in only 6.6 per cent of cases. A definite tuberculous focus was present in 77.3 per cent of his cases of tuberculous fistula.

While opinion is not unanimous, it is generally agreed that a tuberculous anal fistula should be excised. Lockhart-Mummery[110] believes that it is useless to expect healing in a patient who has active tuberculosis in the lungs or elsewhere and advises conservative treatment by establishing satisfactory drainage and sending the patient to a sanatorium. Gabriel[56] recommends operation for a superficial fistula but says that on no account should any extensive operation involving division of the sphincter be performed. Shields[161] advises operation even if the pulmonary lesion is severe. Marino[117] and Gerendasy[60] maintain that removal of the specific focus adds to the patient's comfort and enhances the efficiency of the general treatment of pulmonary tuberculosis. Marino has shown that postoperative healing takes twice as long if the patient has a positive sputum and he believes that the optimum time for operation is when the sputum is negative. He prefers excision with the scalpel to cautery or diathermy excision for cleaner, quicker healing. Gerendasy considers operation indicated even in the presence of a positive sputum. He also prefers operation with the knife if all diseased tissue can be clearly eliminated. Otherwise he prefers the diathermy knife. Hirschman[95] sears the tract thoroughly with the electrocautery or glacial acetic acid. Buie and co-workers[31] have found that healing is definitely slower in tuberculous fistulas. He recommends conservative measures when pulmonary disease is a present menace to life, but even in such patients fistulectomy should be considered when the condition persists in troublesome form. This may sometimes actually save the patient's life. When a patient is not in such an extremely serious condition this authority believes that it is advisable to wait until the pulmonary lesion has become quiescent, but excision should be performed as soon as it is

feasible as a definite aid in overcoming the ravages of pulmonary tuberculosis. Chisholm and Gauss[38] are highly optimistic about surgery, reporting 98 per cent clinically cured in six to twelve weeks in a series of 71 cases.

Attention has been called to the occasional development of malignancy in fistulous tracts by Rosser[155] and others.[33, 52, 101, 106, 113, 129, 137, 191]

Treatment of Anal Abscess.—Delay in operating for anal abscess until fluctuation and pointing occurs is advised by a few.[26, 28, 39] However, many authorities[7, 56, 71, 109] emphatically plead for early wide incision. It is believed that delay allows extension of the suppurative process with consequent delay in healing and the certainty of fistula formation. The hope is held that by early drainage there may be a lesser probability of fistula formation. The diagnosis of early abscess formation by bidigital examination before the development of external evidence of its presence is emphasized by Smith.[170] Operation under gas-oxygen, low spinal or sacral block anesthesia is almost universally advised. Emphasis is placed upon adequate incision for complete, rapid, and continued drainage. The old-time radial incision is poorly devised for this purpose. For subtegumentary and ischiorectal abscesses some advise tangential or T-shaped incision,[193] others suggest always an anteroposterior incision with excision of skin on the lateral border of the incision for continued drainage.[7] Still others[28, 56] employ a cruciform incision, cutting away the skin and subcutaneous tissue between the crossed incisions to leave a roughly circular opening. Packing of the abscess cavity is considered contraindicated and usually a small iodoform wick or rubber drain is inserted for twenty-four to forty-eight hours. Moist heat by wet packs or Sitz baths should be employed as early as is possible. Any attempt to complete the incision, as for a fistulectomy, is discouraged, although this may occasionally be done in small subtegumentary abscesses.[170] Hillemand and co-workers[91] employ x-ray (100 r. two to three times) and claim many cures without fistula formation. The rationale and justification of such a procedure appears extremely doubtful. For submucosal abscesses longitudinal incision through the mucous membrane the entire length of the abscess cavity is universally employed. For pelvirectal abscesses Hibschman[89] objects to the forcing of a clamp through the levator ani muscle after traversing the ischiorectal space, warning of subsequent levator hernia. He has devised an approach made through an anteroposterior midline incision between the anus and the coccyx with division of the coccygeal portion of the external sphincter into two longitudinal bundles, and blunt dissection through the rectococcygeus portion of the levator ani muscle into the retrorectal or lateral pelvirectal spaces.

Treatment of Anal Fistula.—Fistula operation may be more difficult than other rectal operation, not excluding operation for rectal cancer,

according to Lockhart-Mummery.[110] He says that more surgeons' reputations are damaged by unsuccessful operations for fistula than by laparotomy. The bad results of laparotomy are buried with flowers but the fistulas go around the world exhibiting the unsuccessful results of treatment.

Nonsurgical ablation of the fistulous tract by chemicals has been practically completely discarded. The use of a tight ligature or seton to cut through the intervening tissues between the tract and the skin has also been almost wholly abandoned. Recently, however, the use of the elastic ligature for this purpose has been revived by Rochet and Violet.[152] Modification of this procedure using an elastic snare rarely has been employed.[61, 102] At the present time consensus of opinion is overwhelmingly in favor of complete excision of the fistulous tract. The importance of adequate muscle relaxation by suitable anesthesia has already been considered. Sufficient time should elapse after drainage of an acute abscess to allow the abscess cavity to shrink down to a chronic fibrous tract before excision is performed.

The first important step in operation is determination of the site of the primary or internal opening which is in one or another of the anal crypts. In the simpler varieties of fistula careful palpation will often reveal the course of the tract. The actual site of the primary opening may be palpable as a fibrotic depression or may be discernible by an area of deep ulceration or the presence of a small tuft of granulation tissue. Finally, careful exploration of the crypts with a hooked probe will locate the primary opening in the vast majority of cases. Probing of the tract from the external or secondary opening is permissible but this should be done with the most gentle pressure with a blunt probe in order to avoid making false passages or a false internal opening. In the event that these methods fail to localize the primary opening, it is recommended[28] that the tract be laid open from the outside as far as a probe can be easily passed, following which it is usually possible to determine readily the further course of the tract. The use of various dyes (methylene blue, iodine, potassium permanganate, mixtures of methylene blue and hydrogen peroxide) for identification of the tract and of the primary opening is recommended by some.[15, 61, 95, 166, 177] Others[28, 56, 60, 109, 161] definitely disapprove of this practice, claiming that its use may at times mask the true extent of the disease by escaping and staining the surrounding tissue, and insisting that the differentiation between diseased and healthy tissue is more readily made without the interference of artificial coloring. I am in thorough accord with the latter group. In determination of the primary opening some assistance may be gained from "Salmon's Law":[177] When the external opening of a fistula is within 5 cm. of the anus and anterior to a transverse line crossing the center of the anus, the internal opening will be found radially opposite; most fistulas with the external opening posterior to

this line or more than 5 cm. away from the anus anterior to this line have their internal openings at the posterior commissure.

The fundamental principle of operation is the division of all overlying tissue down to the main fistulous tract, excision of the tract, excision of all ramifications of the tract, and adequate excision of skin laterally and distally to assure proper external drainage not only for the time being but until complete healing has taken place. There is still considerable difference of opinion in regard to the problem of division of the sphincter muscles. Complete division is sometimes necessary for the cure of a fistula. There is general agreement that when such division is done a single straight incision should be made at right angles to the fibers of the muscle. It is also essential that the sphincter be cut at only one place. Buie[28] disagrees with both of these principles. Opinions differ concerning division of the muscle at the time of the primary operation. Some authorities believe that this should be done as a second-stage procedure at a later date. Cattell[36] and Buie,[28] both authorities of considerable experience, maintain that two-stage operation is not necessary. They state that if operation is properly done both sphincters can be safely divided and the entire tract excised without loss of sphincter control. The elimination of packing and adequate postoperative care of the wound are considered essential to this success. On the other hand, Lockhart-Mummery[109] advises that the external sphincter never be cut completely at the primary operation but that this be postponed two or three weeks until the muscle is held firmly in the surrounding fibrous tissue so that it will be limited in its retraction after division. He attaches no importance to the internal sphincter in defecatory control and says that this may be cut with impunity. Swinton[177] advocates two-stage operation when all or nearly all of the sphincter must be divided. Gabriel[56] recommends this procedure even when the muscle is to be partially divided. Allen and Haskell[2] report the use of a two-stage procedure in 119 out of a series of 266 cases with good union of the muscle without deformity. In my experience secondary division of the sphincter is very occasionally indicated. This does not depend primarily upon how much of the muscle must be divided but upon how much support remains above and behind the muscle. In the case of a very large, deep cavity in which there is no support for the muscle and healing is to be inevitably a prolonged process immediate division of the sphincter may result in deep fixation of the cut edges of the muscle. Ultimately there then may be a trough or groove extending the length of the anal canal which may interfere with perfect functional closure of the canal. In those cases in which there is no such deep cavity or in which sufficient scar tissue support can be left immediately above the level of the ano-rectal line complete division of the sphincter is done with impunity. In the employment of a two-stage procedure the

entire tract is laid open and excised with the exception of the sphincter muscle. A seton of nonabsorbable material is tied loosely around the muscle. When the underlying cavity has filled in sufficiently to give good fibrous support, the muscle included in the seton is divided by simple incision.

Adequate postoperative care is of great importance. Prolonged packing of the wound interferes with the desired collapse of the walls of the cavity and with optimal healing. All packing should be removed by the third day. Free use of moist heat by hot packs or Sitz baths facilitates cleanliness, diminishes infection, and encourages healing. Nonirritating antiseptic solutions may be used. Exterior drainage must be maintained at all times until the wound finally heals.

Although some authorities advocate the use of the cautery for fistula operation,[166] many believe that it causes unnecessary delay in healing and prefer sharp knife dissection.

ANAL INCONTINENCE

The restoration of function in sphincter incontinence, whether due to damage to the nerve supply or to direct or operative injury to the muscle itself, is a serious problem. To let well enough alone is the frequent and often wise solution for the surgeon. But this answer to the problem is of little comfort to the miserable individual who bears the disability. Surgical repair should never be undertaken lightly. Plastic operations upon the muscle itself are most difficult because of the frequent presence of extensive scar tissue and fibrous degeneration of the muscle. A plastic procedure apparently perfect at the time of operation may be completely nullified by the likely development of infection in the wound.

Wreden[190] and Stone,[175] in 1929, reported a method of reconstruction of voluntary control by the use of double fascial slings passed around the anal canal and harnessed to the gluteus maximus muscle on either side. Stone[176] in 1931 reported additional cases in which a slightly modified procedure was employed. In a total of 11 cases treated by this method 5 were very successful, 4 almost entirely successful, 1 only slightly beneficial, and 1 a complete failure. Voluntary tightening of the interlocking fascial slings was supposedly attained by contraction of the glutei. It would appear more probable that the success of this method of voluntary control is achieved by the upward pull of the anal canal by the levator ani muscles with the lateral ends of the fascial strips held fixed by contraction of the glutei. In such event satisfactory voluntary control is dependent upon the presence of levator function.

Buie[28] considers three principles in operation for postoperative incontinence: (1) eradication of any residual sinuses or abscesses; (2) removal of any deformities, such as hemorrhoids, prolapsing mucosa,

or hypertrophied papillae; (3) excision of all elastic membrane or scar, recreating the cavity which existed immediately following the fistulectomy in such a manner that the postoperative management will produce the proper kind of scar, to which the remnants of the sphincter can adhere and close the rectal outlet.

REFERENCES

1. Abel, A. L.: The Pecten: the Pecten Band: Pectenosis and Pectenotomy, Lancet **1**: 714, 1932.
2. Allen, J. H., and Haskell, B.: A Twostage Operation for Fistula-in-Ano, Surg., Gynec. & Obst. **58**: 651, 1934.
3. Arnheim, E. E.: Hemorrhoidectomy With the High Frequency Electric Current, Am. J. Surg. **41**: 45, 1938.
4. Babcock, W. W.: The Dangers and Disadvantages of Spinal Anesthesia, New York M. J. **98**: 897, 1913.
5. Bacon, H. E.: Anorectal Analgesia With Nupercain (Report of 91 Cases), Current Researches in Anesth. & Analg. **13**: supp. 71, 1934.
6. Bacon, H. E.: Anorectal Fistula, S. Clin. North America **16**: 487, 1936.
7. Bacon, H. E.: Anus, Rectum, Sigmoid Colon—Diagnosis and Treatment, Philadelphia, 1938, J. B. Lippincott Company.
8. Bacon, H. E.: Prolapse and Procidentia of the Rectum: a Reclassification, M. Times, New York **64**: 268, 1936.
9. Balch, F. G.: The Injection Treatment of Internal Hemorrhoids, New England J. Med. **212**: 57, 1935.
10. Ball, Sir Charles: The Rectum, Its Diseases and Developmental Defects, London, 1908, Oxford Press.
11. Bellot, V. J.: Traitement sclérosant en une séance des hémorroides. Résultats d'une expérience de 6 ans, Presse méd. **37**: 1328, 1929.
12. Bensaude, R., and Oury, P.: Traitement des hémorroides par les injections sclérosants. Sept ans d'expérience, Presse méd. **36**: 706, 1928.
13. Bentley, D. F., Jr.: Caudal Anesthesia: Some Observations in Its Use, J. Urol. **18**: 517, 1927.
14. Berry, G. P.: Caudal Epidural Anesthesia in Perineal Surgery of Genito-Urinary Tract: Report of 165 Consecutive Cases, J. A. M. A. **90**: 1018, 1928.
15. Best, R. R.: Preventing the Recurrent Anorectal Fistula, Nebraska M. J. **22**: 288, 1937.
16. Bishop, C. H.: Discussion of Kleckner.[105]
17. Black, W. P.: Use of Galvanism in Hemorrhoids, Tr. Am. Proct. Soc. p. 139, 1934.
18. Blaisdell, P. C.: Pathogenesis of Anal Fissure and Implications as to Treatment, Surg., Gynec. & Obst. **65**: 672, 1937.
19. Blond, K.: Zur pathologie und therapy des verikösen symptomenkomplexes des mastdarms, Wien. klin. Wchnschr. **47**: 1409, 1934.
20. Boas, I.: Eine kleine, aber wichtige verbesserung meiner injektions methode der inneren hämorrhoiden, Deutsche med. Wchnschr. **57**: 225, 1931.
21. Bordier, H.: Un beau résultat du traitement des hémorroides par la diathermo-coagulation, Paris méd. **2**: 229, 1937.
22. Bower, J. O., Wagoner, G., and Clark, J. H.: Clinical and Experimental Investigation in Spinal Anesthesia, Anesth. & Analg. **5**: 95, 1926.
23. Bowman, F. B.: Prolapse of the Rectum, Tr. Am. Proct. Soc. p. 95, 1936.
24. Brenner, I. M.: Proctologic Electrosurgery, Arch. Phys. Therapy **18**: 579, 1927.
25. Brodie, B. C.: Brodie's Pile; Lectures on Diseases of the Rectum; Lecture III, Preternatural Contraction of the Sphincter Ani, M. Classics **2**: 929, 1938.
26. Brust, J. C. M.: Anal Abscess and Anal Fistula; Etiology and Treatment, New York State J. Med. **37**: 400, 1937.
27. Buie, L. A.: Anal Fistulectomy, J. A. M. A. **97**: 1208, 1931.
28. Buie, L. A.: Practical Proctology, Philadelphia, 1937, W. B. Saunders Company.
29. Buie, L. A., and Butsch, W. L.: The Importance of Recognizing Contracted Anus, Am. J. Digest. Dis. & Nutrition **5**: 162, 1938.

30. Buie, L. A., and McCuskey, C. F.: Sacral Anesthesia in Rectal Surgery, Tr. Am. Proct. Soc. p. 19, 1925.
31. Buie, L. A., Smith, N. D., and Jackman, R. J.: The Rôle of Tuberculosis in Anal Fistula, Surg., Gynec. & Obst. **68**: 191, 1939.
32. Burt, C. V., and Rennie, J. G.: Ambulatory Operative Treatment of Anorectal Pathological Conditions; 194 Consecutive Operations Performed Under Local Anesthesia of Prolonged Duration, Surg., Gynec. & Obst. **66**: 1054, 1938.
33. Campbell, J. A.: Fistula in Ano, Multiple External Openings Associated With Malignancy, Tr. Am. Proct. Soc. p. 13, 1930.
34. Carmel, A. G.: Discussion of Green.[70]
35. Cartwright, E. L.: The Management of Strangulated Hemorrhoids, Am. J. Digest. Dis. & Nutrition **4**: 425, 1937.
36. Cattell, R. B.: Recurrent Anorectal Fistulae, S. Clin. North America **15**: 1659, 1935.
37. Chene, P., and Dubarry, J.: Hémorroides et traitement sclérosant, J. de méd. de Bordeaux **112**: 555, 1935.
38. Chisholm, A. J., and Gauss, H.: Anorectal Tuberculosis, J. A. M. A. **104**: 2067, 1935.
39. Christianson, H. W.: Anal Abscess and Anal Fistula, Minnesota Med. **18**: 655, 1935.
40. Coffee, H. D.: Injection Treatment of Prolapsus Ani. Preliminary Report, U. S. Vet. Bur. M. Bull. **6**: 132, 1930.
41. Corning, J. L.: Spinal Anaesthesia and Local Medication of the Cord, New York M. J. **42**: 483, 1885.
42. Daniels, E. A.: Rectal Disorders in Childhood, Am. J. Dis. Child. **54**: 573, 1937.
43. Daniels, E. A.: The Haemorrhoidal Lesion. Its Radical Cure by Submucous Injections With or Without the Ligature Operation, Am. J. Digest. Dis. & Nutrition **2**: 631, 1935.
44. De Takats, G.: Prolongation of Local Anaesthesia, Surg., Gynec. & Obst. **43**: 100, 1926.
45. Dogliotti, A. M.: A New Method of Block Anesthesia. Segmental Peridural Spinal Anesthesia, Am. J. Surg. **20**: 107, 1933.
46. Drueck, C. J.: Cryptitis, Illinois M. J. **71**: 528, 1937.
47. Dunphy, J. E., Alt, R. E., and Reiling, W. A.: Evipal Anesthesia: Clinical Study of 300 Cases, SURGERY **1**: 265, 1937.
48. Edwards, S.: Quoted by Gabriel.[56]
49. Falk, H. C.: Deaths From Spinal Anesthesia, Am. J. Surg. **11**: 461, 1931.
50. Fansler, W. A.: Anal Fissure, Minnesota Med. **20**: 734, 1937.
51. Findlay, L.: Injection Treatment of Complete Rectal Prolapse, Brit. M. J. **2**: 330, 1934.
52. Fitchet, S. M.: Fistula an Etiologic Factor in Rectal Carcinoma, New England J. M. **199**: 766, 1928.
53. Foss, H. L., and Schwalm, L. J.: Relative Merits of Spinal and Ether Anesthesia, J. A. M. A. **101**: 1711, 1933.
54. Frick, J. H.: Present Status of Spinal Anesthesia Based Upon 25,000 Cases, Am. Ther. Soc. **30**: 36, 1930.
55. Gabriel, W. B.: Results of an Experimental and Histological Investigation Into Seventy-Five Cases of Rectal Fistulae, Collected Papers of St. Mark's Hospital, London, 1935, H. K. Lewis & Company, Ltd.
56. Gabriel, W. B.: The Principles and Practice of Rectal Surgery, London, 1932, H. K. Lewis & Company, Ltd.
57. Gabriel, W. B.: Treatment of Pruritus Ani and Anal Fissure; Use of Anaesthetic Solutions in Oil, Brit. M. J. **1**: 1070, 1929.
58. Galbraith, D. A.: A Simple Treatment for Rectal Prolapse, M. J. Australia **2**: 620, 1931.
59. Garside, E.: A Study of Spinal Analgesia Based Upon 357 Personal Cases, Ann. Surg. **94**: 899, 1931.
60. Gerendasy, J.: Tuberculosis of the Anorectal Region, J. M. Soc. New Jersey **35**: 156, 1938.
61. Gorsch, R. V.: Anorectal Fistula. Anatomical Considerations and Treatment, Am. J. Surg. **32**: 302, 1936.
62. Gorsch, R. V.: Discussion of Green.[70]
63. Gorsch, R. V.: Electrosurgery in Hemorrhoids, M. Rec. **144**: 226, 1936.
64. Gorsch, R. V.: Electrosurgery in Proctology, Am. J. Surg. **40**: 436, 1938.

65. Gorsch, R. V.: Physical Measures in Proctology, New York State J. Med. **36**: 1875, 1936.
66. Gorsch, R. V.: Oil Soluble Anesthetics in Proctology, M. Rec. **139**: 35, 1934.
67. Graham, A. B.: The Use of Quinine and Urea Hydrochloride in the Treatment of Fissure in Ano, Tr. Am. Proct. Soc. p. 43, 1926.
68. Graham, G. D.: Desiccation of Hemorrhoids, Arch. Phys. Therapy **16**: 741, 1936.
69. Green, W. O.: Local Anesthesia With Quinine and Urea Hydrochloride—Report of 4 Cases, J. A. M. A. **54**: 1940, 1910.
70. Green, W. W.: Oil-Soluble Anesthetics in Anorectal Surgery. A Clinical Study of 100 Cases With a Control Series of an Equal Number, Tr. Am. Proct. Soc. p. 153, 1937.
71. Green, W. W.: Perirectal Abscesses, Ohio State M. J. **34**: 1245, 1938.
72. Gutiérrez, A.: Anesthesia metamérica epidural, Rev. de cir. de Buenos Aires **11**: 665, 1932.
73. Hall, C. E.: Scientific Management of Anal Fissure, J. M. A. Georgia **25**: 57, 1936.
74. Hanes, G. S.: Discussion of Murrieta.[138]
75. Hanes, G. S.: Rectal Prolapse, Tr. Am. Proct. Soc. p. 134, 1924.
76. Hanes, G. S.: Treatment of Pruritus Ani, Pruritus Vulvae and Rectal Prolapse by Hypodermic Injections of Hydrochloric Acid, Tr. Am. Proct. Soc. p. 112, 1928.
77. Harger, J. R.: Peridural Anesthesia in Abdominal Surgery, Illinois M. J. **65**: 317, 1934.
78. Harpin, R. A.: Caudal Anesthesia in Urology, New England J. Med. **208**: 31, 1933.
79. Harris, H. A.: Discussion on Fistula-in-Ano. Some Embryological Aspects of the Problem Involved, Proc. Roy. Soc. Med. **22**: 43, 1929.
80. Harvey, F.: The Treatment of Hemorrhoids by Galvanism, Clin. Med. & Surg. **42**: 597, 1935.
81. Haskell, B.: Intravenous Anesthetic Agents in Proctology, Tr. Am. Proct. Soc. p. 144, 1937.
82. Haskell, B., and Cheleden, J. J.: Evipal Anesthesia: Its Use in Proctology, Am. J. Surg. **34**: 581, 1936.
83. Haws, M. W.: Anal Fissure and Irritable Anal Ulcer, Kentucky M. J. **35**: 269, 1937.
84. Hayes, H. T.: Benacol in Rectal Conditions: Emphasizing Its Use in Rectal Surgery, South. M. J. **24**: 309, 1931.
85. Heldt, T. J., and Moloney, J. C.: Negative Pressure in Epidural Space. Preliminary Studies, Am. J. M. Sc. **175**: 371, 1928.
86. Herrmann, G., and Desfosses, L.: Sur la muqueuse de la region cloacale du rectum, Compt. rend. Acad. d. sc. **90**: 1301, 1880.
87. Hertzler, A. E.: The Technic of Local Anesthesia, St. Louis, 1933, The C. V. Mosby Company.
88. Hertzler, A. E.: The Use of Diothane in Control of Afterpain in Hemorrhoidectomy, Am. J. Obst. & Gynec. **27**: 301, 1934.
89. Hibschman, H. Z.: Supralevator Abscesses, Tr. Am. Proct. Soc. p. 139, 1937.
90. Hill, T. C., and Hayden, E. P.: Chronic Constipation Due to Congenital Stenosis of the Anal Canal, New England J. Med. **199**: 552, 1928.
91. Hillemand, P., Marchand, J. H., and Bensaude, A.: Traitement ambulatoire des abcès de la region anale par la radiothérapie, Arch. d. mal. de l'app. digestif **28**: 396, 1938.
92. Hiller, R. I.: The Anal Sphincter and the Pathogenesis of Anal Fissure and Fistula, Surg., Gynec. & Obst. **52**: 921, 1931.
93. Hirschman, L. J.: Anorectal Pain and Its Clinical Significance, J. A. M. A. **102**: 348, 1934.
94. Hirschman, L. J.: Perianal Suppuration as a Focus of Infection, J. Michigan M. Soc. **34**: 662, 1935.
95. Hirschman, L. J.: Synopsis of Ano-Rectal Diseases, St. Louis, 1937, The C. V. Mosby Company.
96. Houston, W. R.: Office Treatment of Rectal Disease, Kentucky M. J. **35**: 338, 1937.
97. Howard, C.: Submucous Injection of Haemorrhoids With Quinine Urea Hydrochloride, Lancet **1**: 20, 1929.
98. Jones, H. W.: One Thousand Spinal Anaesthesias, With Special Reference to Complications and Mortality, Ann. Surg. **96**: 85, 1932.

99. Jones, T. E.: Discussion of Kleckner.[105]
100. Jones, W. H.: Spinal Analgesia: a New Method and a New Drug—Percaine, Brit. J. Anaesth. **7**: 99, 1930.
101. Kallet, H. I.: Discussion: Fistula Associated With Malignancy, Tr. Am. Proct. Soc. p. 16, 1930.
102. Katskee, E.: Anorectal Fistulectomy, a New Method, Am. J. Surg. **24**: 105, 1934.
103. Keyes, E. L., and McLellan, A. M.: Preliminary Report on a New Local Anesthetic (Nupercain), Am. J. Surg. **9**: 1, 1930.
104. Kilbourne, N. J.: Local Anesthetics Producing Prolonged Analgesia: Elimination of Pain After Rectal Operations, Surg., Gynec. & Obst. **62**: 590, 1936.
105. Kleckner, M. S.: The Treatment of Hemorrhoids. A Comparison of the Operative and the Nonoperative Methods, Pennsylvania M. J. **38**: 163, 1934.
106. Kraker, D. A.: Discussion: Fistula Associated With Malignancy, Tr. Am. Proct. Soc. p. 15, 1930.
107. Lipschitz, W., and Laubender, W.: Die pharmakoloschen wirkungen des percains: toxikologische untersuchungen, Klin. Wchnschr. **9**: 968, 1930.
108. Lockhart-Mummery, J. P.: Discussion on Fistula-in-Ano, Proc. Roy. Soc. Med. **22**: 1331, 1929.
109. Lockhart-Mummery, J. P.: Diseases of the Rectum and Colon and Their Surgical Treatment, Baltimore, 1934, Wm. Wood & Company.
110. Lockhart-Mummery, J. P.: Fistula-in-Ano, Lancet **1**: 657, 1936.
111. Lundy, J. S.: Intravenous Anesthesia, Am. J. Surg. **34**: 559, 1936.
112. Lundy, J. S.: Intravenous Anesthesia: Preliminary Report of the Use of Two New Thiobarbiturates, Proc. Staff Meet. Mayo Clin. **10**: 536, 1935.
113. Lynch, J., and Gross, P.: A Case of Carcinoma of the Rectum Associated With Fistulae in Ano, Am. J. Cancer **18**: 39, 1933.
114. Magath, T. B., and Feldman, W. H.: The Relative Value of Cultural Methods and Guinea Pig Inoculation in the Diagnosis of Tuberculosis, Am. J. Clin. Path. **2**: 199, 1932.
115. Manheim, S. D., and Marks, M. M.: Eucupin (Isoamylhydrocupreine) as a Local Anesthetic in Proctologic Surgery and in the Treatment of Pruritus Ani, Am. J. Surg. **39**: 86, 1938.
116. Marchand, J. H.: Les agents physiques dans le traitement des hémorroides, Bull. gén. de thérap. **188**: 22, 1937.
117. Marino, A. W. M., Buda, A. M., and Skir, I.: Anorectal Tuberculosis, Tr. Am. Proct. Soc. p. 219, 1938.
118. Martin, C. F.: A Suggested Classification for Anorectal Prolapse, J. A. M. A. **79**: 737, 1922.
119. Martin, E. G.: Prolapse of the Rectum, Its Reclassification and Surgical Treatment, J. A. M. A. **99**: 368, 1932.
120. Mayo, C. W.: Complete Rectal Prolapse—a Fascial Repair, West. J. Surg. **46**: 75, 1938.
121. Mayo, H.: Observations on Injuries and Diseases of the Rectum, London, 1833, Burgess & Hill.
122. McIntyre, W. K.: Discussion of Kleckner.[105]
123. Meisen, V.: Injection Treatment of Haemorrhoids; Survey of 100 Cases, Acta chir. Scandinav. **64**: 311, 1928.
124. Meisen, V.: Varicose Veins and Haemorrhoids and Their Treatment, London, 1932, Levin & Munksgaard.
125. Merar, T. J.: Prevention of Pain Following Hemorrhoidectomy, Am. J. Surg. **43**: 45, 1939.
126. Miles, W. E.: Observations Upon Internal Piles, Surg., Gynec. & Obst. **29**: 497, 1919.
127. Milhiet, H.: Traitement des prolapsus du rectum, Progrès méd. 1092, 1935.
128. Milligan, E. T. C., and Morgan, C. N.: Surgical Anatomy of Anal Canal, With Special Reference to Anorectal Fistulae, Lancet **2**: 1150, 1213, 1934.
129. Moon, L. E.: Development of Carcinomata in Ano-Rectal Fistula Tracts, Tr. Am. Proct. Soc. p. 62, 1926.
130. Moon, L. E.: Discussion of Green.[70]
131. Morgan, C. N.: Oil Soluble Anaesthetics in Rectal Surgery, Brit. M. J. **2**: 938, 1935.
132. Morley, A. S.: Injection Treatment of Complete Rectal Prolapse, With Report of 2 Cases, Brit. M. J. **2**: 204, 1934.

133. Moscowitz, A. V.: The Pathogenesis, Anatomy, and Cure of Prolapse of the Rectum, Surg., Gynec. & Obst. **15**: 7, 1912.
134. Mounsey, J. W.: Diagnosis and Treatment of Anal Fissure, Northwest Med. **36**: 24, 1937.
135. Mühling, A.: Der prolapsus ani et recti und seine behandlung, München. med. Wchnschr. **82**: 1156, 1935.
136. Muller, G. P., and Overholt, R. H.: Spinal Anaesthesia, Ann. Surg. **94**: 738, 1931.
137. Murray, F. H.: Discussion: Relation of Fistula-in-Ano to Cancer of the Anal Canal, Tr. Am. Proct. Soc. p. 71, 1934.
138. Murrieta, A. J.: A New Technique in Correcting Rectal Prolapse, Tr. Am. Proct. Soc. p. 141, 1938.
139. Murrieta, A. J.: Houston Valves as a Factor in Rectal Obstipation, and Other Dysfunctions, With Description of New Operative Technique, Tr. Am. Proct. Soc. p. 160, 1931.
140. Newman, S. E.: The Technic of a Radical Hemorrhoidectomy; a Procedure With Elimination of the Element of Pain, J. Missouri M. A. **34**: 114, 1937.
141. Odom, C. B.: Epidural Anesthesia, Am. J. Surg. **34**: 547, 1936.
142. Pagés, F.: Quoted by Odom.[141]
143. Peyton, T. R.: Significance of Cryptitis and Associated Conditions, M. Rec. **146**: 57, 1937.
144. Potter, E. B., and Wellman, J. M.: Alcohol Injection for Prolapse of Rectum, Am. J. Surg. **19**: 297, 1933.
145. Pruitt, M. C.: Discussion of Green.[70]
146. Pruitt, M. C.: Modern Proctology, St. Louis, 1931, The C. V. Mosby Company.
147. Reuther, T. F.: Caudal Anesthesia in Proctology, Internat. Clin. **3**: 183, 1938.
148. Reuther, T. F.: Duration of Anesthesia With an Oil Soluble Anesthetic Solution, Illinois M. J. **72**: 182, 1937.
149. Riddoch, J. W.: Anal Fissure, Lancet **2**: 1150, 1936.
150. Rider, T. H.: Pharmacological and Preliminary Clinical Observations on Diothane, a New Local Anesthetic, Anesth. & Analg. **12**: 259, 1933.
151. Rider, T. H.: The Testing of Local Anesthetics, J. Pharmacol. & Exper. Therap. **39**: 329, 1930.
152. Rochet, P., and Violet, M.: Treatment of Extrasphincteral Anal Fistulae: Method of Elastic Ligature, Abst. J. A. M. A. **112**: 370, 1939.
153. Rosser, C.: Chemical Rectal Stricture, J. A. M. A. **96**: 1762, 1931.
154. Rosser, C.: Diothane in Surgery of the Anal Canal. A Clinical Survey of 100 Cases, Surg., Gynec. & Obst. **59**: 820, 1934.
155. Rosser, C.: Etiology of Anal Cancer, Am. J. Surg. **11**: 328, 1931.
156. Rosser, C.: Hemorrhoids, Journal-Lancet **53**: 584, 1933.
157. Saphir, J. F.: Ambulant Treatment of Anorectal Diseases, New York M. J. **110**: 709, 1919.
158. Saphir, J. F.: Rectal Operations Under Local Anesthesia, New York M. J. **104**: 644, 1916.
159. Saphir, J. F.: Rectal Operations Under Local Anesthesia, M. Rec. **146**: 398, 1937.
160. Shaw, E. C.: Epidural Anesthesia for Perineal Prostatectomy; an Experimental and Clinical Study With Report of 100 Consecutive Cases, J. Urol. **15**: 219, 1926.
161. Shields, R. M.: Anorectal Fistula, S. Clin. North America **17**: 279, 1937.
162. Sigmond, H. W.: Positive Galvanism for the Treatment of Hemorrhoids, Am. J. Phys. Therapy **3**: 259, 1925.
163. Silvers, H. I.: Hemorrhoids, J. M. Soc. New Jersey **31**: 425, 1934.
164. Simmons, N. J.: Elimination of Postoperative Pain Following Hemorrhoidectomy, New England J. Med. **214**: 20, 1936.
165. Smith, D.: Discussion of Murrieta.[138]
166. Smith, D.: Personal communication.
167. Smith, F. C.: Anorectal Abscess and Fistula, Med. World **54**: 156, 1936.
168. Smith, F. C.: Internal Hemorrhoids, Am. Med. **39**: 2, 1933.
169. Smith, F. C.: The Use of Diothane as a Local Anesthetic in Proctology, M. Rec. **141**: 581, 1935.
170. Smith, N. D.: Acute Anal Pain From Obscure Abscesses: Their Diagnosis and Treatment, New England J. Med. **215**: 195, 1936.
171. Smith, N. D.: Hemorrhoidectomy: A Plastic Operation, Minnesota Med. **20**: 233, 1937.

172. Smith, T. E.: Anal and Perianal Fissures and Ulcers, Dallas M. J. **24**: 33, 1938.
173. Speare, G. S., and Mabrey, R. E.: Anal Fissure—Evaluation of Treatment With Oil Injections, New England J. Med. **219**: 302, 1938.
174. Steinberg, N.: Recent Advances in the Treatment of Rectal Diseases by Injection Methods in Ambulatory Patients. I. The Use of Gabriel's Modified Solution in the Treatment of Fissure-in-Ano, New England J. Med. **213**: 162, 1935.
175. Stone, H. B.: Plastic Operation for Anal Incontinence, Arch. Surg. **18**: 845, 1929.
176. Stone, H. B.: Plastic Operation to Restore Voluntary Anal Control, J. A. M. A. **97**: 1205, 1931.
177. Swinton, N. W.: Anorectal Fistulae, S. Clin. North America **17**: 871, 1937.
178. Sykes, W. S.: Modern Treatment-Anaesthesia. With a Section on Local Anaesthesia by P. J. Moir, New York, 1932, W. W. Norton & Company, Inc.
179. Terrell, E. H.: Discussion of Murrieta.[138]
180. Terrell, E. H.: The Treatment of Hemorrhoids, Med. World **54**: 293, 1936.
181. Terrell, E. H.: The Treatment of Hemorrhoids by a New Method, Tr. Am. Proct. Soc. p. 65, 1916.
182. Tucker, C. C., and Hellwig, C. A.: Anal Ducts, Comparative and Developmental Histology, Arch. Surg. **31**: 521, 1935.
183. Uhlmann, F.: Über percain, ein neues lokalanesthetikum, Narkose u. anaesth. **2**: 168, 1929.
184. Warshaw, D.: Surgery of Rectal Diseases by Electrothermic Methods—Based on 1,100 Cases, Am. J. Surg. **41**: 437, 1938.
185. Webb, C. H.: Congenital Malformations of the Rectum and Anus as a Cause of Constipation, New Orleans M. & S. J. **90**: 457, 1938.
186. Weese, H., and Scharpff, W.: Evipan, ein neuartiges einschlafmittel, Deutsche med. Wchnschr. **58**: 1205, 1932.
187. Weiss, D.: Ambulatory Treatment for Anal Fissure, Am. J. Surg. **37**: 88, 1937.
188. Werner, J.: Quoted by Bacon.[7]
189. Woolf, M.: Personal communication.
190. Wreden, R. R.: A Method of Reconstructing a Voluntary Sphincter Ani, Arch. Surg. **18**: 841, 1929.
191. Yeomans, F. C. Discussion: Fistula Associated With Malignancy, Tr. Am. Proct. Soc. p. 14, 1930.
192. Yeomans, F. C.: Fissure of the Anus and Thrombotic Hemorrhoids, Am. J. Surg. **6**: 426, 1929.
193. Yeomans, F. C.: Proctology: A Treatise on the Malformations, Injuries and Diseases of the Rectum, Anus and Pelvic Colon, New York, 1929, D. Appleton & Company.
194. Yeomans, F. C., Gorsch, R. V., and Mathesheimer, J. L.: Benacol in the Treatment of Pruritus Ani, Tr. Am. Proct. Soc. p. 24, 1927.
195. Zobel, A.: Personal communication.

Review of Recent Meetings

REPORT OF THE MEETING OF THE SOCIETY FOR EXPERIMENTAL PATHOLOGY, APRIL 26-29, 1939, TORONTO, ONTARIO

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(From the Department of Pathology, Harvard Medical School)

AT THE Toronto meeting of the Society for Experimental Pathology (April 26 to 29), a number of important papers were presented. Only a few of them can be enumerated here.

G. A. Bennett and **M. Shaffer**, Boston, Mass., reported an interesting study on the passage of foreign proteins from the circulating blood into joints and other body cavities, including the anterior chamber of the eye and the spinal cord cavity. These workers succeeded in demonstrating a differential permeability to proteins in the various body cavities. From the blood stream a detectable concentration of egg albumen or horse serum accumulated into joints. The concentration in the aqueous humor was definitely lower. In the majority of instances none of these proteins reached the spinal fluid. They interpret their results as a possible explanation for the higher protein concentration of synovial fluid than that of spinal fluid or of aqueous humor. It is perhaps questionable whether their findings explain the high protein concentration of long-standing joint effusions, inasmuch as inflammatory exudates, in general, reveal a high concentration of proteins owing to increased capillary permeability. These investigators likewise pointed out, curiously enough, that, whereas egg albumen was rapidly excreted through the kidney, horse serum failed usually to appear in the urine. This raises various questions pertaining to differential renal function. Their investigations with pneumococci Type III likewise add further support to the findings with proteins, indicating again the high physiologic permeability of synovial structures for elements contained in the circulating blood. These studies throw considerable light on the comparative permeability of several important body cavities.

D. H. Sprunt and **S. McDearman**, Durham, N. C., reported studies showing an increased resistance to infection with vaccinia by the administration of estrogenic hormone in castrated or pseudopregnant female rabbits.

H. L. Stewart and **B. F. Jones**, Cambridge, Mass., described the curious occurrence of a spontaneous disease involving the cecum in rats. The condition is virtually an ulcerative cecitis with involvement of adjacent lymph nodes. The lymph stasis which they described perhaps may be referable in part to the conspicuous fibrinous deposits in distended lymph channels, inducing thus a partial lymphatic blockade.

Owen H. Wangensteen and **Clarence Dennis**, Minneapolis, Minn., presented an extremely interesting concept on the obstructive origin of appendicitis in man. Their observations seem to indicate that the vermiform appendix secretes fluid.

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The secretion pressure of this fluid may reach heights approaching that of the systolic level of blood pressure. The temporary obstruction resulting from such fluid secretion may contribute to the histopathologic picture of acute appendicitis. Such an interpretation is highly instructive. It is now well known that inflammatory edema is probably largely referable to the establishment of an effective lymphatic blockade that prevents the rapid diffusion of excessive fluid, which in turn has permeated from vascular channels. The increase in secretory activity of the diseased appendix, by inducing a rise in pressure, perhaps can temporarily interfere with the local capillary filtration equilibrium. This would be conducive to anoxemia and inflammation. A knowledge of the mechanism of this increased fluid secretion would be valuable in an endeavor to elucidate further the pathogenesis of appendicitis.

D. R. Coman, Philadelphia, Pa., reported some interesting observations on chemotaxis by studying the nature of substances attracting or repelling the slime mold. The behavior of this mold, *Physarum polycephalum,* resembles that of an enormous leucocyte which manifests both positive and negative chemotactic reactions. Owing to its size, it can readily be studied with the naked eye. Coman found that this organism was invariably repelled by HCl and NaOH at definite concentrations. The effect was entirely referable to the H and OH ions. On the other hand, dextrose induced positive chemotaxis; whereas, insulin or crystallized egg albumen was indifferent. The conclusions drawn refer chemotaxis to a subtle selective reaction rather than to mere pH concentration or surface activity of a substance. The contributory part of this work seems to be the introduction of a new convenient tool in the form of a large visible organism which seems, nevertheless, to be similar in its behavior to a leucocyte, but which is evidently considerably simpler for the study of a complex biological property.

Finally, **V. Menkin,** Boston, Mass., reported observations concerning the mechanism of leucocytosis with inflammation. He was able to demonstrate the presence of a leucocytosis-promoting factor in inflammatory exudates. Leucotoxin induces increased capillary permeability and leucocytic migration into injured tissue; but it fails to alter the level of leucocytes in the blood stream. The leucocytosis-promoting factor induces a prompt rise in the leucocyte counts of otherwise normal dogs. This factor is thermolabile and essentially nondialyzable.

REPORT OF THE MEETING OF THE AMERICAN ASSOCIATION FOR CANCER RESEARCH, APRIL 5 AND 6, 1939 RICHMOND, VA.

E. T. Bell, M.D., Minneapolis, Minn.

(From the Department of Pathology, University of Minnesota Medical School)

1. **R. Schrek,** Hines, Ill.: **The Innate and Clinical Malignancy of Cutaneous Carcinomas.**—Basal cell carcinomas have a much longer clinical duration than squamous cell carcinomas of the skin. In a large group there was a 20 per cent fatality from squamous cell tumors and much lower death rate from the basal cell growths.

2. **Lawrence Berman** (by invitation) and **E. T. Bell,** Minneapolis, Minn.: **Extragenital Chorioepithelioma.**—Chorionepithelioma was reported in which there

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were tumors in the lungs, liver, and brain, but no tumor in any of the pelvic organs. The uterus, tubes, and ovaries were examined carefully by sections about 3 mm. in thickness. No primary growth could be found. The uterus showed a very marked decidual reaction, such as occurs in ectopic pregnancy. There was luteinization of the ovarian follicles. Pregnancy cells were demonstrable in the hypophysis. Fourteen months before her death the patient had given birth to a normal child. Following this pregnancy, there was intermittent bleeding of small amount for ten months. During the last four months of her life, she had symptoms largely referable to a tumor of the brain.

3. **Lawrence W. Smith,** Philadelphia, Pa.: **Effect of Reduced Temperatures on Carcinoma.**—Smith was able to produce striking regression of superficial tumors by prolonged application of cold to the tumor. Also, in two cases of generalized metastases he obtained striking relief of pain and regression of the tumors by repeated lowering of the body temperature of the patient down to a rectal temperature of 95°. One patient who had generalized metastases from a carcinoma of the breast was apparently well two years later. The improvement in these two cases was very impressive.

4. **Egon Lorenz** (by invitation) and **Harold L. Stewart** (by invitation), Washington, D. C.: **Effect of Long-Continued Feeding of 1:2:5:6 Dibenzanthracene to Mice.**—Long-continued feeding of dibenzanthracene to mice produced no tumors in the intestinal tract, and only an occasional tumor of the lung developed. Studies of the various organs indicated that the hydrocarbon was not absorbed from the intestine.

5. **Michael Levine,** New York, N. Y.: **The Cytology of 1:2:5:6 Dibenzanthracene Mouse Tumors.**—The author transplanted dibenzanthracene tumors in one instance for fourteen generations and in another for twenty-two generations. The tumors at first were fibrosarcomas for several generations and then changed to a foam cell type of tumor. In later generations they returned to the fibroblastic structure. The author thought these variations were due to accidental selection of certain tumor cells in the transplant.

6. **Arnold M. Seligman** (by invitation) and **M. J. Shear** (by invitation), Cambridge, Mass.: **Studies in Carcinogenesis. VIII. Experimental Production of Brain Tumors in Mice With Methylcholanthrene.**—The authors implanted small pellets of methylcholanthrene in the cerebral cortex of mice. They obtained thirteen tumors after 200 days. A large variety of brain tumors was represented: fibrosarcoma, spongioblastoma, oligodendroglioma, ependymoma, and neuroepithelioma. The various types were illustrated convincingly by lantern slides.

7. **Ira T. Nathanson** and **Darrel T. Shaw** (by invitation), Boston, Mass.: **The Effect of Testosterone Propionate on the Development and Growth of Spontaneous Mammary Carcinoma in Female Mice.**—By injections of testosterone the authors were able to decrease the incidence of spontaneous carcinoma of the breast in female mice, but testosterone had no effect on tumors already established.

8. **Wilton R. Earle** (by invitation) and **Carl Voegtlin,** Washington, D. C.: **Further Observations on the Mode of Action of Methylcholanthrene on Cultures of Normal Tissues.**—Methylcholanthrene added to cultures of normal tissues caused some suppression of the rate of growth.

9. **George Gey** (by invitation), **Margaret R. Lewis** (by invitation), and **Margaret K. Gey** (by invitation), Baltimore, Md.: **Continuous Cultures of Blood Cell Tumors and Their Transplantability.**—Leucemic blood cells are maintained satisfactorily in tissue cultures. The culture was transplantable at any time

and would produce leucemia in susceptible animals. A moving picture was shown illustrating the movements of lymphoblasts and monocytes in the cultures. They were unable to maintain a continuous culture of normal leucocytes, but with the malignant blood cells they were successful.

10. **F. N. Craig** (by invitation), **J. M. Muus** (by invitation), and **W. T. Salter,** Boston, Mass.: **The Metabolism of Mouse Sarcomas 180 Growing at Different Rates.**—The metabolism of slowly growing and rapidly growing mouse sarcomas was studied. There was no difference in the consumption of lactic acid, but less oxygen was used by the slowly growing tumors. The metabolism failed to reflect the rate of growth accurately.

11. **Arthur L. Amolsch** (by invitation), Detroit, Mich.: **Mixed Mesodermal Tumors of the Female Genitals.**—The author demonstrated polypoid uterine tumors with a very complex structure. The tumors showed areas of myxoma, areas of cartilage, and areas of rhabdomyoma.

12. **Shields Warren,** Boston, Mass.: **The Prognostic Value of the Response to Radiation in Carcinoma of the Cervix.**—A series of cases of carcinoma of the cervix were treated by radiation and biopsies were taken from time to time to determine the effect of the radiation. The biopsies showed that some tumors did not respond to radiotherapy. It was advised that such growths be removed surgically. The histologic grade of the tumor had no relation to its radiosensitivity.

13. **Isabel M. Scharnagel,** New York, N. Y.: **Newer Methods in Differential Diagnosis of Mammary Tumors.**—The author claimed the correct diagnosis of 119 tumors of the breast by x-ray examination. She recommended x-ray as a valuable means of distinguishing benign from malignant tumors of the breast.

14. **Kenneth M. Lynch,** Charleston, S. C.: **Carcinoma of Lung in Asbestos Silicosis.**—In 35 cases of asbestosis of the lungs the author found 2 primary carcinomas of the lung. In the literature these tumors are reported as being mainly of the squamous cell type but some are glandular.

15. **J. S. McCartney,** Minneapolis, Minn.: **Case of Granulosa Cell Carcinoma of Ovary.**—In a patient 57 years of age a granulosa cell tumor of the ovary was removed six months after the onset of symptoms. Seven years later, when the patient was 64 years of age, there was a recurrence of the tumor and the patient died of metastases. Post-mortem examination showed metastases in the peritoneum, lymph nodes, and brain.

16. **Wm. J. Hoffman,** New York, N. Y.: **Hormone Therapy of Male Breast Hypertrophy.**—In several cases of gynecomastia the author was able to produce regression of the breast by treatment with testosterone. He admitted that the breast often undergoes spontaneous decrease in size, but he thought the treatment hastened this regression.

17. **G. B. Mider** and **J. J. Morton,** Rochester, N. Y.: **Skin Tumors of Mice Following a Single Application of Methylcholanthrene.**—Malignant tumors of the skin of mice were obtained after a single application of methylcholanthrene. Papillomas appeared at the site of application in thirty-one to forty-eight days. Three mice developed carcinomas of the skin in 121 and 248 days after the application; 40 per cent of the mice developed papillomas.

18. **Kanematsu Sugiura,** New York: **Observations on Animals Painted With Tobacco Tar.**—Tobacco tar failed to produce cancer of the skin in mice.

19. **S. G. Warner** and **M. C. Reinhard**, Buffalo: **The Effect of X-ray on a Transplantable Tumor of Known Genetic Constitution.**—A tumor, originating spontaneously in a stock of brown mice, grew in about 100 per cent of transplants in mice of the same strain but failed to grow when transplanted into the C57 black strain. However, when the spontaneous tumor was radiated with doses of 50 to 100 r., it could then be transplanted successfully into the C57 strain. The authors thought that they had produced a genetic change in the tumor.

20. **Warren H. Lewis**, Baltimore, Md.: **Normal and Malignant Cells.**—Moving pictures were shown illustrating the differences in the mobility of various forms of malignant cells.

21. **Louis F. Fieser** (by invitation), Cambridge, Mass.: **Chemical Studies of Carcinogenic Hydrocarbons.**—Fieser discussed the chemical structure of the various carcinogenic hydrocarbons. He stated that he had new methods of synthesizing these hydrocarbons and that some of these, notably benzpyrene, were available commercially. Efforts are being made to conjugate hydrocarbons to protein in the hope of producing antigenic substances. Shifting of the methyl group changes the carcinogenic activity of the hydrocarbon. A large number of new compounds were being experimented with.

22. **Wm. S. Murray**, Buffalo, N. Y.: **Is the Transmission of Mammary Cancer Susceptibility Mendelian in Character?**—Murray pointed out that it is very difficult to get a strain of mice entirely free from carcinoma of the breast. He had the C57 black strain which at first gave no cancers but now gives a few. Reciprocal crosses of the low cancer strain with a high cancer of the breast strain gave twice as many cancers when the mother was from the high cancer stock. By back-crossing continuously with a male parent of the high cancer strain, he finally obtained mice in which he computed that the chromatin was practically the same as that of the original parent of high cancer stock. However, these mice obtained in this way had a very low incidence of cancer of the breast. He concluded that mammary cancer is not transmitted through the chromosomes and is therefore not Mendelian.

23. **John J. Bittner**, Bar Harbor, Me.: **The Possible Method of Transmission of Breast Cancer Susceptibility in Mice.**—Bittner gave the results of further studies of the frequency of cancer in mice of a high cancer stock nursed by low cancer mothers. When the mice of the high cancer strain were taken immediately after birth and fostered by the low cancer mother, the incidence of cancer was only 1 per cent. When mice of the low cancer stock were fostered by a high cancer mother, the incidence of cancer was 11 per cent. He thinks, therefore, that the milk is a very strong influence in the development of cancer of the breast in mice.

24. **Halsey J. Bagg** and **John Jackson** (by invitation), New York, N. Y.: **A Cancer Line in Mice Derived From Low Tumor Strain (C57 Black).**—Bagg, in an experiment carried on over a period of four years, has developed a strain of mice from the low cancer strain (C57) in which 37 per cent of the breeding females developed cancer. This strain was obtained by selection from mice that developed spontaneous cancer of the breast.

25. **C. P. Rhoads** (by invitation), New York, N. Y.: **The Natural Defense Mechanism Against Experimental Carcinogenesis.**—Rhoads believes that the relative insusceptibility of the rabbit and the rat to dibenzanthracene is due to their greater ability to convert this hydrocarbon into a harmless substance.

26. **L. G. Rowntree** (by invitation), **Arthur Steinberg** (by invitation), and **W. R. Brown** (by invitation), Philadelphia, Pa.: **A Further Report on the Production of Sarcomas in Rats by the Feeding of Crude Ether Extracted Wheat-Germ Oil.**

Arthur Steinberg (by invitation), **W. R. Brown** (by invitation), and **L. G. Rowntree** (by invitation), Philadelphia, Pa.: **Pathology of Masses Produced in Rats by the Feeding of Crude Ether Extracted Wheat-Germ Oil.**

W. R. Brown (by invitation), **Arthur Steinberg** (by invitation), and **L. G. Rowntree** (by invitation), Philadelphia, Pa.: **Factors Influencing the Formation of Tumors Produced by Crude Ether Extracted Wheat-Germ Oil.**—Rowntree and his associates reported the results of their work with wheat-germ oil. They were able to obtain a large number of sarcomas in rats by oral administration of wheat-germ oil. A great many of the tumors produced had been studied microscopically and most of them were reported as spindle cell sarcomas. However, it was not clear how many of the masses produced were true tumors and how many were inflammatory masses. On the whole, the evidence presented was not convincing. Several who took part in the discussion stated that they had been unable to produce sarcomas in rats by means of wheat-germ oil.

REPORT OF THE MEETING OF THE AMERICAN ASSOCIATION OF PATHOLOGISTS AND BACTERIOLOGISTS, APRIL 6 AND 7, 1939, RICHMOND, VA.

E. T. Bell, M.D., Minneapolis, Minn.

(From the Department of Pathology, University of Minnesota Medical School)

1. **Herbert S. Reichle** and (by invitation) **John L. Work**, Cleveland, Ohio: **The Incidence and Significance of Healed Miliary Tubercles in the Liver, Spleen, and Kidneys.**—In 452 autopsies the author found 91 cases with small calcified nodules in the spleen, liver, and kidneys. These had no relation to blood vessels. Fifty per cent of the cases had tuberculosis, active or healed, in other parts of the body. In 3 of the nodules tubercle bacilli were demonstrated. Interpretation of these small miliary nodules is that they represent healed tuberculosis.

2. **William H. Feldman** and (by invitation) **A. H. Baggenstoss**, Rochester, Minn.: **The Occurrence of Virulent Tubercle Bacilli in Presumably Nontuberculous Tissues of the Lung.**—Some years ago Opie reported the finding of living tubercle bacilli in normal lung tissue. Feldman took samples from normal lung tissue, studied a portion microscopically, and injected other portions into guinea pigs. Sixteen guinea pigs were inoculated from each post mortem. Only 3 positive growths were obtained in the guinea pigs and these were from 2 individuals. The author, therefore, concludes that living tubercle bacilli are not found in normal lung tissue.

3. **Otto Saphir**, Chicago, Ill.: **Intracystic Papilloma of the Breast.**—Saphir studied fifty-eight papillomas of large ducts of the breast, most of which were associated with bleeding from the nipple. Some of the tumors were studied in serial sections. He described pseudoglandular and glandular microscopic varieties. There was also a form composed of spindle-shaped cells which he called transitional cell type. All of the papillomas are apparently benign, but the author advised mastectomy in the case of the transitional cell papillomas.

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4. **Samuel A. Goldberg,** Newark, N. J.: **Splenic Neoplasms.**—Several varieties of primary neoplasms of the spleen were described, including lymphangioma, hemangioma, lymphosarcoma, and endothelioma. The author believes that metastatic tumors do not grow readily in the spleen, since he believes the splenic tissue is antagonistic to the growth of carcinoma. In the discussion, it was pointed out that metastases are even less frequent in the kidneys than in the spleen and that, therefore, the spleen probably does not exert any antagonistic influence to the growth of carcinoma.

5. **Rigney D'Aunoy, Bjarne Pearson** (by invitation), and **Béla Halpert,** New Orleans, La.: **Carcinoma of the Lung. An Analysis of 74 Necropsies.**—The authors found 74 primary carcinomas of the lung in 6,600 post-mortem examinations. In their experience carcinoma of the lung was about one-half as frequent as carcinoma of the stomach. Sixteen of the tumors were columnar cell type; 37, squamous cell carcinomas; 21, reserve cell carcinomas (apparently the authors were calling the ordinary round cell carcinoma a reserve cell type). In the discussion it was pointed out that the high incidence of primary carcinoma of the lung was probably due in part to selection in autopsies. It was held that carcinoma of the stomach occurs more than twice as frequently as primary carcinoma of the lung.

6. **Amour F. Liber** and **James R. Lisa,** New York, N. Y.: **Stromal Tumors of the Choroid Plexus.**—Two types of tumors originating in the choroid plexus were described. One growth was the ordinary papilloma, the other the stromal cell tumor. The authors paid special attention to the tumors developing from stromal cells in the plexus.

7. **Gordon Ritchie,** Madison, Wis.: **Metastatic Tumors of the Myocardium.**—In 3,000 post-mortem examinations a large variety of metastatic tumors were found in the myocardium. Some of the tumors reached the myocardium by way of the blood stream, others by the lymph stream, and still others by direct extension. No primary myocardial tumors were seen. No clinical symptoms were attributed to the growths in the myocardium.

8. **Walter Schiller** (by invitation), Chicago, Ill.: **Concepts of a New Classification of Ovarian Tumors.**—Schiller proposed a new classification of ovarian neoplasms based largely on embryology. He would distinguish one group which arises from structures within the ovary, such as fibromas, granulosa cell tumors, leiomyomas, angiomas, and luteinomas; and a second group, which develops from structures displaced into the ovary during development, such as the mesonephric tumors. He also recognized that tumors, such as the endometriomas, originated in adult life from grafting of endometrium on the ovary. In the discussion it was pointed out by Ewing that this classification has no advantages over those commonly in use, since the histologic structure of the ovarian neoplasm is frequently not characteristic of any known type. Regardless of what classification one uses, tumors will be encountered which are exceedingly difficult to diagnose accurately.

9. **Herbert L. Ratcliffe,** Philadelphia, Pa.: **Carcinoma of the Kidney in a Colony of Rhesus Monkeys.**—In a family of rhesus monkeys consisting of seventeen members, one parent and three offspring had hypernephromas of the kidney. The histologic structure is very similar to the ordinary hypernephromas in human beings.

10. **Harry S. N. Greene,** Princeton, N. J.: **The Pathology of Mammary Carcinoma of the Rabbit.**—Primary carcinomas of the breast in the rabbit were

discussed. Two distinct morphologic types were recognized. In one form the cancer develops in a previously cystic breast. In the other form no previous cystic disease exists. These tumors often produce endocrine changes. The pituitary may be four to five times the normal size due to hyperplasia of the chromophobe cells. The endometrium frequently shows cystic hypertrophy which suggests an excess of estrin. Some of the carcinomas were transplanted successfully.

11. **Roland S. Aronson (by invitation)**, Philadelphia, Pa.: **The Skins of Guinea Pigs After Five Years' Daily Applications of 1:2:5:6 Dibenzanthracene.**—Aronson applied dibenzanthracene daily to the skins of guinea pigs for five years. He obtained thickening of the epidermis and hyperplasia, but no carcinomas developed.

12. **Hugh G. Grady** (by invitation) and **Harold L. Stewart,** Cambridge, Mass.: **Histologic Study of the Development of Pulmonary Tumors Induced by 1:2:5:6 Dibenzanthracene and Methylcholanthrene in Strain A Mice.**—By subcutaneous injections of dibenzanthracene and methylcholanthrene in strain A mice the authors secured a very high percentage of multiple carcinomas of the lungs. These developed in 90 per cent of the injected mice over a period of twenty-six to sixty days. Multiple primary tumors were demonstrated. The tumors seemed to arise from the alveolar wall. The authors did not commit themselves as to the exact cell types which gave rise to the tumors. The metastases sometimes assumed the appearance of a spindle-cell sarcoma.

13. **Robert A. Moore** and (by invitation) **Allister McLellan,** New York, N. Y.: **The Effect of Combined Androgen and Estrogen on the Prostate.**—Estrogen causes benign hypertrophy of the prostate in the rat. Testosterone suppresses the sexual cycle of the female rat. Fifty units of androgen are required to neutralize one unit of estrogen. Daily intramuscular injections of both hormones produced the same changes as estrin alone.

Papers 14 to 20 composed a **Symposium on Hypertension**:

14. **Harry Goldblatt,** Cleveland, Ohio: **Experimental Observations on the Pathology and Pathogenesis of Essential Hypertension.**—At the invitation of the Council, Dr. Goldblatt gave a summary of his work on experimental hypertension. As is well known, Dr. Goldblatt produces hypertension in dogs by constriction of the renal arteries on both sides. Under moderate constriction, the dogs develop a marked rise of blood pressure which persists indefinitely. Some animals have been maintained with a hypertension for as long as six years. Under moderate constriction there is no marked disturbance of renal function, but the urea clearance is decreased. This corresponds to clinical benign hypertension. When the renal arteries are severely constricted, a disease develops in the dog which Goldblatt considers similar to malignant hypertension in man. The animals develop uremia and show hemorrhages in the eye grounds. There are also occasional arteries in the intestine and elsewhere which show the arteritis characteristic of malignant hypertension. Goldblatt believes that primary hypertension in man is due to disease of either the arterioles or the arteries in the kidneys.

No hypertension results from clamping the splenic, femoral, or celiac arteries. In those instances in which the collateral circulation develops after clamping the renal arteries, the hypertension disappears. The experiments indicate that hypertension is due to ischemia of the kidneys which results in the formation of a pressor substance. In one type of experiment Goldblatt ligatured one ureter and constricted the other renal artery. He found a necrotizing arteriolitis in the kidney with the obstructed ureter. Hypertension occurs when the kidneys are denervated and

is therefore not of reflex origin. In the dog with experimental hypertension the blood pressure falls gradually if the adrenals are removed. No elevation occurs in an animal if the arteries are constricted after removal of the adrenals. He thinks that a portion of adrenal cortex is necessary to maintain the hypertension. Goldblatt takes the position that primary hypertension is basically a disease of the vascular system of the kidneys.

15. **C. C. Erickson** and **L. V. Dill** (by invitation), Durham, N. C.: **Observations on the Effects of Renal Ischemia in Pregnant Dogs and Rabbits.**—In pregnant dogs the authors produced bilateral constriction of the renal arteries by the Goldblatt method. The dogs rapidly developed toxic symptoms similar to human eclampsia. At post-mortem examination hemorrhagic necroses of the liver were found. The changes in the kidneys were not clearly brought out. Emptying of the uterus produced relief of symptoms and survival of the animal.

In the discussion Goldblatt stated that if one first produces the constriction of the renal arteries and then allows the animals to become pregnant, the blood pressure tends to decrease as pregnancy advances. Greene spoke of spontaneous eclampsia in rabbits.

16. **Hugo A. Freund,** Detroit, Mich.: **Vascular Hypertension Occurring in Cases of Coarctation of the Aorta.**—Freund described five clinical cases of coarctation of the aorta. The blood pressure is always high in the upper extremities unless the heart fails. In one case the author recorded a diastolic pressure of 110 in the legs. The systolic pressure in the lower extremities is low, but the diastolic is as high as in the upper extremities. He agrees with Rytand that the hypertension in coarctation of the aorta is due to ischemia of the kidneys. In the discussion Goldblatt agreed with this interpretation.

17. **F. W. Hartman,** Detroit, Mich.: **Hypertension and Kidney Lesions Produced by X-ray.**—Hartman called attention to the fact that he had produced experimental hypertension in dogs fourteen years ago by means of radiation of the kidneys. He repeated this earlier work and described the arteriolosclerosis which results from radiation of the kidneys. He stated that by this method of producing hypertension one also has arteriolosclerosis of the kidneys.

18. **Irvine H. Page,** Indianapolis, Ind. (by invitation), and **Irving Graef,** New York, N. Y.: **Hypertension Following Experimental Perinephritis Induced by Cellophane. A Preliminary Report.**—Page enclosed the kidneys in a cellophane membrane. This results in a fibrinopurulent exudate on the capsule of the kidneys. The exudate becomes organized and finally results in marked compression of the kidney, including the renal vein. The tubules undergo atrophy. He thinks that the chief cause of the atrophy is compression of the renal vein. The animals develop hypertension after this procedure. Hypertension was attributed to renal ischemia.

19. **Robert Brotchner** (by invitation) and **E. T. Bell,** Minneapolis, Minn.: **Experimental Acute Hypertension From Obstruction of the Aorta.**—The authors produced acute hypertension by stenosis and occlusion of the thoracic aorta in dogs. The blood pressure was recorded by means of a cannula in the carotid artery. Stenosis of the aorta above the origin of the celiac axis produces an average rise of 56 mm. in the systolic pressure. When the constriction is applied immediately above the kidneys, a rise of about 14 mm. takes place. With the clamp below the kidneys, the rise is only about 8 mm. Stenosis or occlusion of both renal arteries did not affect the blood pressure. It is noteworthy that the degree of hypertension corresponds with the amount of the blood that is obstructed. Ob-

struction above the celiac axis occludes about 60 per cent of the blood flow, while obstruction immediately above the kidneys blocks only about 30 per cent of the blood flow. The rise of blood pressure is immediate and, therefore, cannot be due to any pressor substance from the kidneys. Also, occlusion of the renal arteries does not cause a rise of blood pressure. The authors, therefore, interpret this type of acute hypertension as due to mechanical obstruction of the blood flow. Similarly, they consider that the hypertension in the upper extremities in coaretation of the aorta is due to mechanical interference with the blood flow and not to ischemia of the kidneys.

20. **H. Edward MacMahon,** Boston, Mass.: **Vascular Measurements in Hypertension.**—MacMahon measured the cross-sectional area of arteries in normal persons and in those with permanent hypertension. He noted that in chronic hypertension there was usually dilatation of the larger arteries in the kidney rather than constriction.

21. **Morton McCutcheon** and (by invitation) **O. Tod Mallery, Jr.,** Philadelphia, Pa.: **Motility and Chemotaxis of Leucocytes in Health and Disease.**—The authors studied the motility and chemotaxis of leucocytes from normal persons and from diseased individuals. They found that in persons acutely ill the rate of movement of the leucocytes was slower than normal, but the rate of approach to bacteria on the slide was about the same. In other words, the leucocytes from the diseased person followed a more direct route toward the bacteria.

22. **P. B. Parsons, Mary A. Poston,** and **Bowman Wise** (by invitation), Durham, N. C.: **The Pathology of Human Brucellosis.**—The authors made cultures of lymph nodes from ten different patients in whom the microscopic picture of the lymph node was that of typical Hodgkin's disease. In eight of these ten cases they grew pure cultures of brucella. They considered that these patients had brucellosis and not Hodgkin's disease. Two of their patients had died. (It appears probable that the authors were dealing with Hodgkin's disease in persons who had a latent brucellosis. The clinical course of the disease corresponds to Hodgkin's disease. If the authors make a subsequent report in a few years, they will probably find that all of their patients are dead. It is not to be expected that brucellosis will have a high mortality.)

23. **Charles F. Branch** and **Sidney Farber,** Boston, Mass.: **The Pathology of Equine Encephalitis in Man.**—A group of cases of equine encephalitis in man was reported. The authors had seen seventeen cases at autopsy. The disease affected mainly infants and children and had a 95 per cent mortality. It is an acute fulminating encephalitis. Histologically the meninges show serum, fibrin, lymphocytes, and neutrophiles. The arteries show perivascular cuffs and sometimes an acute arteritis. There is no sharp histologic separation from the St. Louis type of encephalitis.

24. **E. A. Linell,** Toronto, Ontario: **The Significance and Importance of Hemorrhage Into the Brain Stem in Cases of Head Injury.**—The author reported 21 cases with hemorrhage in the brain stem following injuries to the head. There were 6 subependemal hemorrhages, 8 in the upper midbrain, 7 in the lower midbrain. He pointed out that brain-stem hemorrhages may occur without fracture of the skull.

25. **W. L. Robinson,** Toronto, Ontario: **A Study of Bronchiectasis in 50 Lobectomy Cases.**—Robinson reported a histologic study of fifty lobectomies for bronchiectasis. No specific organisms were obtained by culture. In the dilated bronchi the epithelium is intact. There are only occasional ulcers. Bronchiectasis

is, therefore, not an ulcerative bronchitis but an inflammation in the subepithelial tissues of the bronchus. Apparently the stagnation of the bronchial secretion is the major cause of the persistence of the infection. In one case there was an adenoma of the bronchus.

26. **J. S. McCartney,** Minneapolis, Minn.: **Age and Site of Operation in Relation to Postoperative Pulmonary Embolism.**—McCartney showed from an extensive study that pulmonary embolism occurs much more frequently in old persons than in the young. The reason why certain operations are more often followed by pulmonary embolism than others is due to the age of the patients on whom such operations are performed. For example, operations on the prostate are nearly always performed on old men, and this is the main reason why pulmonary embolism is so frequent. On the other hand, appendectomies are more often performed on younger persons and have a lower incidence of pulmonary embolism. It was shown rather clearly that the age of the patient is more important than the site of the operation in the occurrence of pulmonary embolism.

27. **Warren C. Corwin** (by invitation), Philadelphia, Pa.: **The Relationship of the Insulin Hypoglycemic Reaction to Shock.**—Corwin gave evidence that the hypoglycemic reaction is not related to the phenomena of shock.

28. **Sheldon A. Jacobson,** Brooklyn, N. Y.: **Studies in Metaplastic Ossification.**—Jacobson pointed out that if portions of the urinary bladder of the dog are transplanted into the rectus muscle, bone and cartilage develop from the transplant. When this operation is performed in rats, however, very few positive results are obtained. In the guinea pig autoplastic transplants of the bladder frequently resulted in the production of bone. By experimental procedures, the author was unable to influence the frequency of this osseous transformation.

29. **Edwin F. Hirsch** and (by invitation) **Russell H. Morgan,** Chicago, Ill.: **The Causal Significance to Traumatic Ossification of the Fibrocartilage in Tendon Insertions.**—Hirsch showed that fibrocartilage is present in the tendons at their bony attachments and that in myositis ossificans the formation of bone is due to a primary growth of this fibrocartilage out into the tendon of the muscle.

REPORT ON THE MEETINGS OF THE AMERICAN SOCIETY OF BIOLOGICAL CHEMISTS, APRIL 26-29, 1939, TORONTO, ONTARIO

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OF THE 160 papers presented in person or by title, those abstracted below are considered to be of interest to clinicians.

E. J. Baumann and **D. B. Sprinson,** Montefiore Hospital, New York, N. Y., reported thyroid deficiency in rabbits to result in an increase in the amount of circulating parathyroid hormone as demonstrated by the Hamilton and Schwartz test.

W. R. Bloor, University of Rochester, N. Y., described an improved method for the determination of adrenalin in blood. The results are accurate to 5 per cent and normal blood was found to contain 0.05 μg. adrenalin per cubic centimeter.

Erwin Chargoff, College of Physicians and Surgeons, Columbia University, New York, N. Y., used radioactive phosphorus to study the rate of formation of phospholipids and found lecithin to be formed faster than cephalin in the intestinal tract and liver.

T. B. Coolidge, Duke University School of Medicine, Durham, N. C., found the type of van den Bergh reaction is not affected by the cholic acid content of blood and that the function of alcohol in the indirect test is purely catalytic.

E. Brand, B. Kossel, and **M. Heidelberger,** College of Physicians and Surgeons, Columbia University, New York, N. Y., applied Bergmann's theory of protein structure to thyroglobulin (molecular weight 675,000) and calculated that each molecule contains 5,760 amino acid residues, of which 10 are diiodotyrosine and 2 thyroxine.

D. V. Frost and **C. A. Elvehjem,** University of Wisconsin, Madison, Wis., found that small amounts of cobalt are required, along with iron and copper, for complete regeneration of hemoglobin in anemic dogs maintained on a milk diet.

R. B. Gibson, J. Findlayson, and **W. D. Paul,** State University of Iowa, Iowa City, Ia., using improved methods and procedure, have confirmed the old observation that the renal threshold for glucose is definitely lowered in late pregnancy.

O. M. Helmer and **Irvine Page,** Lilly Laboratory for Clinical Research, Indianapolis, Ind., have made considerable progress in the purification of rennin, the substance responsible for the hypertension of renal ischemic origin, and find it to have the properties of a protein.

Ancel Keys and **H. L. Friedell,** Departments of Physiology and Radiology, University of Minnesota, Minneapolis, Minn., have adapted roentgenkymography to the accurate measurement of the stroke output of the heart. The method has the advantage that it can be applied to persons incapable of cooperation. In mitral insufficiency and aortic regurgitation a comparison of the result obtained with the new method with that of the acetylene (Grollman) method gives a

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quantitative measure of the leak. Roentgenkymography has been found by these workers to be of great value in the diagnosis of adhesive pericarditis and in an estimation of the improvement in function resulting from pericardiolysis.

B. F. Miller, Z. Baker, and **M. J. C. Allinson,** Department of Medicine, University of Chicago, Chicago, Ill., employed the method of Miller and Dubos to distinguish between true and apparent creatine and creatinine in tissues. Except for muscle, brain, and testis, the true creatine is only 15 to 70 per cent of the apparent creatine.

A. F. Morgan, J. Hendricks, and **N. Shimotori,** University of California, Berkeley, Calif., found a low calcium adequate phosphorus diet to prevent the onset of symptoms of hypervitaminosis D_2 and D_3 in rats.

J. Orten and **Aline Orten,** Wayne University, College of Medicine, Detroit, Mich., reported that a long-time protein restriction in a dietary that was otherwise adequate resulted in the development of a typical hypochromic anemia in dogs.

L. B. Pett, University of Alberta, Edmonton, Alberta, examined 1,300 persons, 1,000 of whom were university students, and found that 24 per cent exhibited signs of marked hypovitaminosis A.

H. Schneider and **H. Steenback,** University of Wisconsin, Madison, Wis., found a low phosphorus diet to result in the formation of calcium citrate urinary calculi. Some human urinary calculi were found to contain calcium citrate and it was suggested that this substance may play a role in the formation of the primary nidus upon which other urinary salts are precipitated.

E. Sobel and **B. Sobel,** Jewish Hospital, Brooklyn, N. Y., demonstrated a method for calcium determination in 0.1 c.c. of serum.

A. Sobel, H. Yuska, D. D. Petrovsky, and **B. Kramer,** Jewish Hospital, Brooklyn, N. Y., found a low calcium high phosphorus diet to be most effective in maintaining a low blood lead content in animals fed a constant amount of lead. The effects of vitamin D on the blood lead varied with the type of diet, but the lead in the bone ash was increased by giving this vitamin with diets of varying calcium and phosphorus contents.

Book Reviews

A Manual of Fractures and Dislocations. By Barbara Bartlett Stimson, College of Physicians and Surgeons, Columbia University, New York City. Paper. Pp. 214, with 95 illustrations. Philadelphia, 1939, Lea and Febiger. $2.75.

This little manual summarizes well the whole field of fractures and dislocations. In the preface the author states that the handbook is intended primarily for medical students, but it could be read with profit by the general surgeon. The methods of diagnosis and treatment are those carried out by the staff of the Fracture Service at the Columbia-Presbyterian Medical Center. No attempt has been made to make this an exhaustive treatise. However, it is surprising how comprehensively the various types of fractures are reviewed in such a few pages; it is doubtful whether there is any more essential information to be added. The summaries given at the end of each section are excellent. The illustrations are clear. An index is appended. Without doubt this is one of the best outlines on the subject of fractures published to date.

Oh, Doctor! My Feet! By Dudley J. Morton. Cloth. Pp. 116, with 11 illustrations. New York, 1939, D. Appleton-Century Company. $1.50.

The volume represents an effort on the part of the author to popularize his views on foot trouble. Dr. Morton has studied the foot and written on this subject before. His viewpoint as to the cause of foot trouble and best method of treatment has already quite widespread recognition among orthopedic surgeons.

Whether this effort to popularize the author's ideas will accomplish its purpose remains to be seen. It does graphically describe his theories and method of treatment. Whether or not treatment of foot ailments can be simplified to the extent that the author implies in this work remains to be proved. There are some useful hints as to proper shoe fitting and altogether the book deserves much attention, perhaps more among physicians than among laymen who may be misled by the implied simplicity of diagnosis and cure of all foot ailments.

The Treatment of Fractures. By Charles L. Scudder. Ed. 11. Pp. 1208, with 1717 illustrations. Philadelphia, 1938, W. B. Saunders Company.

In this volume the author has had the assistance of twenty collaborators in writing fifteen of the forty-seven chapters. A successful attempt has been made to include discussion of the various subjects closely allied to fractures. For example, discussion on dangers and precautions in roentgenologic treatment, and aspects of medicolegal relations, though brief, are included.

In the reviewer's opinion, one of the most serious defects lies in the lack of a bibliography. Only at the end of a few chapters are complete references, including recent publications, found. Throughout the book numerous references to

names are included but the scarcity of journal references is so pronounced as to be discouraging to a reader who might want to read further on some particular subject.

Compound fractures are very thoroughly discussed but it is doubtful if most surgeons would agree to the use of such meticulous care in Carrel-Dakin therapy as is presented. Factors in the pathogenesis of shock are clearly presented but a more formal and detailed description of therapeutic measures would be appropriate. The technique involved in the various steps of operations on fractures is carefully presented and illustrated. The chapters dealing with head injuries, anesthesia, fractures of facial bones, and fractures of the vertebral column are particularly well written and informative. Although many neurosurgeons would disagree with the suggestion that hypertonic solutions have but a small place in the treatment of head injuries, attention is correctly called to this possible source of disagreement. The reviewer was gratified to find the statement that wood splints were perhaps the most effective type of the various agents used in the fixation of Colles' fractures.

The insertion of Figs. 1412 and 1413 illustrating fracture of the thigh in the chapter dealing with fractures of the leg is confusing to the reader because of the difficulty in correlating them with the text, particularly with the legends accompanying them.

There is a generous number of illustrations, which consist of an appropriate ratio of photographs, drawings, and photomicrographs. They are extremely well chosen and reproduced; the fact that a great number of them are borrowed does not, of course, detract from their value.

In general, the material covers the subject of fractures very well and includes a great mass of information. The attempt to make the text practical has been extremely successful. It may conscientiously be recommended highly to the student or practitioner for a guide in fracture work.

Midwifery. By ten teachers. Cloth. Pp. 676, with 200 illustrations and 9 color plates. Baltimore, 1938, William Wood and Company. $6.

This book, written by ten teachers and edited by Sir Comyns Berkeley, Clifford White, and Frank Cook, has been prepared primarily for students but is also a very good book for the man in practice. The illustrations are numerous and are very descriptive but the photography does not compare with that in American textbooks.

The description and illustrations for early pregnancy are very clear. The physiologic changes induced by pregnancy have not been described adequately. In the future editions this chapter should be revised thoroughly.

Under the term toxemia the authors consider toxemia, hyperemesis, and acute yellow atrophy. The description of these conditions as well as those of the toxemias is entirely from the clinical point of view. There is very little physiology presented. There seems to be confusion in regard to the various types of liver pathology occurring in eclampsia and hyperemesis. Oliguria and anuria of eclampsia are treated by decapsulation. No mention is made of hypertonic glucose solution which has eliminated anuria in toxemic patients. Medical management for 36 to 48 hours and then induction of labor by rupture of the membranes also is advised. This seems a rather long time to treat patients before inducing labor.

Anemia is treated with iron, yeast, and green vegetables.

Deliveries are usually in the lateral position, which, of course, is rarely used in this country. If packing is used in the treatment of placenta previa, only vaginal packing is used. This is contrary to the teaching that it is most important to pack the lower uterine segment; thus always a portion of the placenta is separated. The use of dettol cream on the vulva and on the pack is mentioned. This antiseptic solution is used very extensively in the British Isles but has not been well received in this country.

The classical and low cervical types of cesarean section are described. In the latter operation the fetal head is delivered with the Willet's forceps. This is far superior to the use of the volsellum forceps. Sterilization is opposed unless there are definite medical grounds for it.

The book is well written and is recommended by the reviewer.

Surgical Pathology of the Diseases of the Mouth and Jaws. By A. E. Hertzler. Cloth. Pp. 248, with 206 illustrations. Philadelphia, 1938, J. B. Lippincott Company. $5.

This book, by the author's own admission, is the last of a series of ten monographs on various phases of surgical pathology. The scope of the work is somewhat more restricted than its title indicates. The writer has discussed in a rather individual style those conditions with which he has become acquainted through the years of an extensive country practice. As a record of experience in which the writer frequently has included his personal ideas concerning the proper diagnostic and therapeutic procedure, the book is interesting and perhaps worth while. For this very reason, however, it cannot be recommended to students as a textbook or to the practitioner as a reference book. Appended at the end of each chapter is a representative bibliography covering the material presented. Just how much value one is to attach to these references is doubtful, for in his preface the writer states that he has not read these references, but on the other hand has "adopted the more respected practice" of having his secretary copy them out of the cumulative index.

By far the most worth-while feature of the book is the wealth of excellent photographs which it contains. Most of the well-known pathologic lesions of the mouth are clearly depicted in their various phases. A number of good photomicrographs of removed specimens further embellish the book.